ASSESSMENT OF TMJ MORPHOLOGICAL CHANGES IN RHEUMATOID ARTHRITIS PATIENTS USING CONE BEAM COMPUTED TOMOGRAPHY

Dissertation Submitted to

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

For partial fulfillment of the requirements for the degree of

MASTER OF DENTAL SURGERY

BRANCH – IX

ORAL MEDICINE AND RADIOLOGY

THE TAMILNADU DR. MGR MEDICAL UNIVERSITY
CHENNAI – 600 032

2014 – 2017

CERTIFICATE BY THE GUIDE

This is to certify that **Dr. PRIYANKA**, Post graduate student (2014-2017) in the Department of Oral Medicine and Radiology (**Branch IX)**, Tamilnadu Government Dental College and Hospital, Chennai 600003, has done this dissertation titled "**ASSESSMENT OF TMJ MORPHOLOGICAL CHANGES IN RHEUMATOID ARTHRITIS PATIENTS USING CONE BEAM COMPUTED TOMOGRAPHY"** under my direct guidance and supervision in partial fulfillment of the M.D.S. degree examination in April 2017 as per the regulations laid down by Tamilnadu Dr. M.G.R. Medical University, Chennai- 600 032 for M.D.S., Oral Medicine and Radiology (Branch – IX) degree examination.

Prof. Dr. S. JAYACHANDRAN, M.D.S, Ph.D. MAMS, MBA

Professor and Head of Department,

Department of Oral Medicine and Radiology,

Tamilnadu Government Dental College and Hospital,

Chennai -600 003.

CERTIFICATE BY HEAD OF THE DEPARTMENT /

HEAD OF THE INSTITUTION

This is to certify that the Dissertation entitled "**ASSESSMENT OF TMJ MORPHOLOGICAL CHANGES IN RHEUMATOID ARTHRITIS PATIENTS USING CONE BEAM COMPUTED TOMOGRAPHY"** is a bonafide work done by **Dr. PRIYANKA,** Post Graduate student (2014-2017) in the Department of Oral Medicine and Radiology under the guidance of **Prof. Dr. S. JAYACHANDRAN, M.D.S., Ph.D.** Professor, Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital, Chennai – 600 003.

Dr. S. JAYACHANDRAN, M.D.S., Ph.D., MAMS., MBA.,
Professor and Head of the Department,
Department of Oral Medicine and Radiology,
Tamil Nadu Government Dental College & Hospital,
Chennai - 600 003.

Dr. B SARAVANAN M.D.S., Ph.D.,
PRINCIPAL
Tamil Nadu Government Dental College & Hospital,
Chennai - 600 003.

DECLARATION BY THE CANDIDATE

Title of the study	**"Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography"**
Place of study	**TamilNadu Government Dental College and Hospital, Chennai 600003**
Duration of the course	**Three years**
Name of the Guide	**Prof. Dr. S. JAYACHANDRAN, MDS, PhD, MAMS, MBA.**
Head of the Department	**Prof. Dr. S. JAYACHANDRAN, MDS, PhD, MAMS, MBA.**

I, **Dr PRIYANKA** hereby declare that no part of the dissertation will be utilized for gaining financial assistance/any promotion without obtaining prior permission of the Principal, TamilNadu Government Dental College and Hospital, Chennai 600003. In addition, I declare that no part of this work will be published either in print or in electronic media without the guide who has been actively involved in the dissertation. The author reserves the right to publish the work with the prior permission of the Principal and Guide, TamilNadu Government Dental College & Hospital, Chennai 600003.

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ACKNOWLEDGEMENT

With supreme sincerity, obedience, deep sense of gratitude and heartfelt appreciation, I thank my esteemed guide, **DR. S. JAYACHANDRAN M.D.S., PhD., MAMS.** Professor and Head, Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital, Chennai – 3, for his valuable guidance, support and encouragement throughout my post graduate course and to bring this dissertation to a successful completion.

I extend my special thanks to our Principal **Dr. B Saravanan, MDS, Ph.D, TNGDCH** for providing motivation and encouraging environment to conduct this study.

I sincerely thank **Dr. G.V. MURALI GOPIKA MANOHARAN M.D.S.,** Professor Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital, Chennai – 3, for his valuable guidance and sincere support.

My sincere humble regards and gratitude to **Dr. L. Kayal M.D.S.,** Professor, **Dr. Capt. P. Regu M.D.S., Dr. K. Sarala, M.D.S., Dr. Vidya Jayaram**, **Dr. Aarthi Nisha**, Assistant Professors, Department of Oral Medicine and Radiology for their help and suggestions during my course

I dedicate this work to my parents **Mrs. SAROJ KHOBRE and Mr. SHAMSHER SINGH KHOBRE,** my brother **RAKESH SINGH KHOBRE**, my sister **NEHA PARVEEN GUNPAL** and family members for their love, care, kindness and prayers, without whom I wouldn't have achieved anything in my life.

I am very much thankful to my uncle **Dr. DINESH KATARIA** and my friends **Dr. NEHA AZAD** and **Dr. JAYAVARMA R** for their suggestions, support and help throughout this study.

I am very much thankful to Department of rheumatology, RGGGH, for kind cooperation and support throughout my study.

I take this opportunity to express my gratitude to my **seniors, batchmates, juniors and friends** for their valuable help and suggestions throughout my course. I also want to thank my department radiographers for their help during my study.

Lastly, I wholeheartedly thank the **ALMIGHTY** who has showered his blessings from above and who is behind each and every step in my life.

LIST OF ABBREVIATIONS USED

RA	Rheumatoid arthritis
CBCT	Cone beam computed tomography
CT	Computed tomography
TMJ	Temporomandibular joint
TMD	Temporomandibular disorder
3D	Three dimensional
RF	Rheumatoid arthritis
ANA	Antinuclear antibody
ESR	Erythrocyte sedimentation rate
CRP	C reactive protein
MTP	Metatarsophalangeal joint
MCP	Metacarpophalangeal joint
PIP	Proximal interphalangeal joint
ACPA	Anti-citrullinated protein antibodies
TNF	Tumor necrosis factor
MRI	Magnetic resonance imaging
RANKL	Receptor activator of nuclear factor kappa b, ligand
ACR	American College of Rheumatology
SNP	Single-nucleotide polymorphism
TNFR	Tumor necrosis factor receptor
EBV	Epstein barr virus

DNA	Deoxy-ribonucleic acid
HLA	Human leukocyte antigen
AKA	Anti-keratin antibodies
APF	Anti-perinuclear factor
SE	Shared epitope
IL	Interleukin
FLS	Fibroblast-like synoviocytes
EULAR	European League Against Rheumatism
DMARD	Disease-modifying anti-rheumatic drugs
CCP	Cyclic citrullinated peptide
OPG	Orthopantomogram

CONTENTS

LIST OF PHOTOGRAPHS

LIST OF TABLES

LIST OF CHARTS

ABSTRACT

TITLE- Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography

BACKGROUND- Rheumatoid arthritis is an autoimmune disorder, which can cause temporomandibular joint involvement with clinical symptoms such as pain, swelling, crepitation and movement impairment. Erosion of the components of TMJ is seen in two-third of the patients with RA. Other radiologic features for RA patients with TMJ include flattening, sclerosis, subchondral cyst and osteophytes. CBCT found to be efficient for diagnosing several osseous changes in the TMJ region.

AIM- The aim of the study is to evaluate osseous changes in rheumatoid arthritis patients with temporomandibular joint involvement (TMD) using Cone Beam Computed Tomography.

OBJECTIVES- To interpret the CBCT axial, coronal, sagittal and 3D images of involved condyle, glenoid fossa and joint space in rheumatoid arthritis patients, in relation to the osseous changes and to correlate the clinical findings with CBCT imaging features.

METHODOLOGY- Patients were diagnosed with rheumatoid arthritis based on laboratory values for rheumatoid factor antibody and anti-citrullinated protein antibodies. A clinical diagnosis of TMJ RA was made based on symptoms and CBCT for each patient involving 100 TMJ was taken.

RESULTS- When the clinical findings of pain at rest and motion at left TMJ was

compared to radiographic feature of osteophyte at left condyle ($p < 0.05$), showed statistically significant values. Crepitus heard at left TMJ was correlated to left condyle bony changes for presence of osteophyte results in $p < 0.05$ in coronal view. While crepitation at right TMJ, assessed for sclerotic changes in opposite side (left) of condyle, results in p value of 0.04 in axial, 0.01 in coronal and 0.00 in sagittal showed highly significant result.

Conclusion- Presence of osteophyte was found to be significant in sagittal section of CBCT at left condylar surface, in subjects with no pain at rest and motion at left condyle. Also, Clinical finding of crepitation is linked to increase evidence of osteophyte, and coronal view should be section of choice to assess this finding. Crepitus heard in one joint can be a reason for sclerotic changes in other joint.

Key words- Rheumatoid arthritis, Temporomandibular joint, Erosion, Osteophyte, Sclerosis, Cone beam computed tomography

INTRODUCTION

Rheumatoid arthritis is a chronic autoimmune condition characterized by inflammation of synovial membrane, destruction of articular surface and sub articular bone, autoantibody production and systemic features including skeletal, pulmonary, cardiovascular and psychological disorders[1,2]. It is considered multifactorial, with combination of strong genetic factors and infections, associated with the incidence of RA. Rarely trauma, acute injury and surgeries are known to initiate RA[3]. RA has 1% prevalence worldwide, with female predilection (3:1). It mainly affects 35 to 55 years of age group. 50% of the RA patients usually presents with TMJ complaints. Clinical symptoms incudes dull, deep aching pain at preauricular region, morning stiffness of joint, joint sound and limited range of motion[1,4]. Hematologically, patients with RA have certain abnormal blood antibodies like “RF” and “ANA” and raised ESR values. Other tests for confirmation like anti- citrulline antibody and CRP test, are required in doubtful cases. Diagnosis of RA with TMJ involvement is based on history, clinical finding, laboratory values and radiographic findings[3]. Erosion of the components of TMJ is seen in two-third of the patients with RA[1]. Evidence for the link between articular erosion and autoimmunity was revealed by anti-carbamylated protein antibodies and anti-citrullinated protein antibodies (ACPA) in the serum of patients with RA. ACPA binds to the surface of the cell and increases differentiation of cell to bone-resorbing osteoclasts, reflecting osteoclast-mediated bone resorption[5]. Both systemic osteoporosis and erosions is associated with imbalance between the osteoclasts and osteoblasts activity. Cytokines related to RA physiopathology, such as RANKL and tumor necrosis factor alpha (TNF-α), are also associated with the pathogenesis of focal and systemic bone lesions[6].

Histopathology of synovial tissue, showed a very large quantity of osteoclasts in bone erosions in RA patients, and less or even absence of mature osteoblasts, suggesting the blocking of differentiation of bone forming osteoblasts by certain molecules in the synovium[5]. Radiological examination is an important tool to assess TMJ disorder[3]. Various imaging techniques for diagnosing RA include extraoral radiography, magnetic resonance imaging, Computed Tomography, arthrography and arthrotomography.

More recently, CBCT is the best-recommended imaging techniques for TMJ evaluation[3]. However, as compared to conventional dental radio- graphic techniques, CBCT gives increased radiation doses to the patients. The introduction of cone beam CT (CBCT) provides a radical change in dental and maxillofacial radiology. Diagnosis of wide range of clinical applications can be improved potentially by three-dimensional (3D) information offered by CBCT, that too at lower doses than seen in ‘‘medical’’ multislice CT[7]. Hintze et al compared conventional tomography with cone beam computed tomography in detection of TMJ morphological changes. They found no significant differences in diagnostic accuracy between the two techniques. Since there is no evidence that CBCT is inferior to CT, the use of imaging technique depends mainly on the available equipment[8]. The dose of radiation in CBCT is lower and is less costly; the amount of radiation differs according to the brand of the equipment, and the anatomical structure to be imaged. Other than these advantages, CBCT also provides a three-dimensional image of the mineralized tissue in maxillofacial region with minimal distortion. CBCT found to be efficient for diagnosing several osseous changes in the TMJ region[9]. Destruction at the TMJ in patients having RA is correlated to the duration and severity of the disease[3]. CBCT can remarkably find out osseous changes at TMJ such as

erosion of articular and subarticular surface of bone, flattening of condylar head, reduction in the joint space width, subchondral cyst and resorption. Joint space reduction is due to the destruction of disc by the pannus[1]. Glenoid fossa also can show erosion and flattening of the articular eminence. Synovial proliferation is the initial process in RA and can differentiate it from other types of arthritis, and is seen readily on MRI. Joint effusion is also more common in RA. As the disease progresses, it can lead to resorption of the mandibular condyle[4]. Management of inflammation and pain is by the use of first-line drugs, such as cortisone and aspirin. Reducing the progression of the disease and promoting remission is by using second-line drugs (disease-modifying anti-rheumatic drugs), such as methotrexate and hydroxychloroquine[1].

AIM

The aim of the study is to evaluate osseous changes in rheumatoid arthritis patients with temporomandibular joint involvement (TMD) using Cone Beam Computed Tomography.

OBJECTIVES

1. To interpret the CBCT axial, coronal, sagittal and 3D images of involved condyle of the mandible in rheumatoid arthritis patients, in relation to the morphological characteristics like erosion, flattening, sclerosis, subchondral cyst and osteophyte.
2. To interpret the CBCT axial, coronal, sagittal and 3D images of involved glenoid fossa in rheumatoid arthritis patients, in relation to the morphological characteristics like erosion, flattening and sclerosis.
3. To interpret the CBCT axial, coronal and sagittal images of involved joint space in rheumatoid arthritis patients, in relation to the morphological characteristics like normal joint space and decreased joint space.
4. To correlate the clinical findings with CBCT imaging features.
5. To formulate a diagnostic algorithm based on the imaging features.

REVIEW OF LITERATURE

Historical background

The first description of RA is found in Augustin Jacob Landre-Beauvais dissertation, which was acknowledged by modern medicine from the year 1800. He treated patients with severe joint pain, which was not explained at the time by other known maladies. He hypothesized that these patients were suffering from a previously uncharacterized condition, which he named Goutte Asthenique Primitive, or "Primary Asthenic Gout."

Alfred Garrod, an English physician, was next important contributor during the mid to late 19th century was the first, who differentiated gout from other arthritic condition.

Alfred Garrod in 1859 wrote "Treatise on Nature of Gout and Rheumatic Gout". He distinguished arthritis from gout and described RA as a distinct condition, which he referred to as "Rheumatic Gout."

In the late 19th and early 20th centuries, two preliminary paleopathological studies were carried out independently by Professor Flinders Petrie and Sir Armand Ruffer. They demonstrate skeletal damage similar to RA of human remains from Egypt.

Archibald Garrod, who was the fourth son of Alfred Garrod, in 1890, wrote the extensive Treatise on Rheumatism and Rheumatoid Arthritis. In this book, he coined the term "Rheumatoid Arthritis".

Arcini, in 1992 described the most striking case of RA in paleopathological samples. The pattern of damage to the right hand, from a human remains found in Europe, showing both damage of the index finger and ulnar deviation, was most likely the case of RA[10].

Published paleopathological studies of Rheumatoid Arthritis[10].

Date of Samples	**Year Published**	**Location Hypothesis Supported**
4000-1000 BCE	1917	Egypt Ancient Origin
3000-1000 BCE	1988	Alabama New World to Old World
2750-2625 BCE	1897	Egypt Ancient Origin
2500-1900 BCE	1988	Sweden Ancient Origin
2290-2040 BCE	1990	Kentucky New World to Old World
1300 BCE	1940	Lower Egypt Ancient Origin
800 BCE	1988	Ohio New World to Old World
400-1 BCE	1985	Denmark Ancient Origin
339-210 BCE	1979	Sicily Ancient Origin
100 BCE	1983	England Ancient Origin

During 2750-2625 B.C, examination of 50-year old Egyptian male mummy showed typical changes of rheumatoid arthritis (RA) in the temporomandibular joint and joints of the hand[11].

EPIDEMIOLOGY

Linos A et al (1980)[12] conducted study of the incidence and prevalence of rheumatoid arthritis in Rochester from 1950-74, which revealed an average annual incidence rate of 28.1/100,000 for males, 65.8/100,000 females. Age-specific rates generally increased with age. Prevalence rates for January 1, 1975 were 4.0/1000 for males and 10/1000 for females. Among adults, prevalence rates were 5.8/1000 for males and 13.4 for females.

A cross-sectional study of clinical evaluations for 4800 new patients of all ages, admitted to a hospital with rheumatological symptoms, in the eastern state of West Bengal performed by **Kar et al (1991)[13]** showed a prevalence of 5.2% of RA among these patients .

Another study to estimate the prevalence of rheumatoid arthritis in adult Indian population by **Malaviya et al (1993)[14],** showed in 299 individuals diagnosed for RA, a prevalence of 0.75% was found. The prevalence of RA in India is quite similar to that reported from the developed countries. It is higher than that reported from China, Indonesia, Philippines and rural Africa. These findings are in keeping with the fact that the north Indian population is genetically closer to the Caucasians than to other ethnic groups.

A study by **Symmons DP et al (1994)**[15] provides the first data on the incidence of RA based on a prospective population-based register. Out of 210 patients, 104 were diagnosed for RA at the time of presentation. The annual incidence rate was 36/100,000 for women and 14/100,000 for men. RA was rare in men aged under 45 yr. The incidence in men rose steeply with age. The incidence in women rose up to age 45 yr, plateaued to age 75 yr, and fell in the very elderly.

A population based cohort study by **Gabriel SE et al (1999)**[16] described trends in the epidemiology of RA over a period of 30 years. Of the 425 individuals, with a mean age at diagnosis of 60.2 years, the overall prevalence of RA was approximately 1%. This incidence was approximately double in women compared with that in men, and increased steadily with age, until age 85, after which the incidence of RA decreased.

An estimation of the global burden of RA, was carried out by **Marita Cross** et al **(2000)**[17]. A series of systematic reviews were conducted to gather age-sex-specific epidemiological data for RA prevalence, incidence and mortality. The global prevalence of RA was found as 0.24%. Globally, of the 291 conditions studied, RA was ranked as the 42nd highest contributor to global disability, just below malaria and just above iodine deficiency.

There is wide variation in the occurrence of RA among countries and areas of the world. **Alamanos et al (2006)**[18], analysed a total of 28 studies based on ACR criteria, across four categories: North America, northern Europe, southern Europe and developing countries.

Involvement of temporomandibular joint in RA, was studied by **Kurtoglu C et al (2016)[19],** Fifty-four patients having RA treatment were examined to evaluate the prevalence and type of temporomandibular disorders (TMD) in patients with rheumatoid arthritis(RA), the prevalence of TMD in RA patients was 90.7%.

ETIOLOGY

No single cause has been identified for RA. It appears to be a multifactorial disease in which there are important genetic and environmental influences[3].

MacGregor A et al (2000)[20] analyzed data from 2 previously published nationwide studies of twins with RA, conducted in Finland and the United Kingdom. Genetic factors have a substantial contribution to RA in the population, accounting for approximately 60% of the variation in liability to disease. Although tempered by power considerations, there is no evidence in these twin data that the overall genetic contribution to RA differs by sex, age, age at disease onset, and disease severity.

A study was conducted by **Barton A et al (2001)[21]** where 291 individuals genotyped for A/G polymorphism in exon 1 of TNFRI and also typed for a single-nucleotide polymorphism (SNP) in exon 6 of the TNFRII gene. The results of this study provided evidence of association between a single-nucleotide polymorphism (SNP) in the TNFRII gene and RA, the strongest association being observed in patients with a family history. No evidence of association between RA and TNFRI was demonstrated.

In evaluation of the environmental associations with rheumatoid arthritis**, Alspaugh MA et al (1978)[22]** first reported an animal model of EBV-induced arthritis and strongly suggested a causative role of the virus in RA.

A study by **Jobanputra P et al (1995)**[23] established association with parvovirus infection, wherein, of the 23 patients with B19 DNA, 29% of patients were diagnosed for rheumatoid arthritis.

Smoking has an adverse effect on disease progression in patients with RA. In a study by **B. Masdottir et al (2000)**[24]**,** sixty-three individual with advanced RA answered a structured questionnaire that included detailed information about their smoking history. They were also evaluated clinically and radiologically. An association was observed between smoking, and those RF types that predispose to RA and have the highest diagnostic specificity for this disease.

Martti Oka (1953)[25] concluded that the onset of rheumatoid arthritis during pregnancy or immediately after it, is so common that in certain circumstances, pregnancy can be regarded as an aetiological factor.

PATHOGENESIS

Historically, RA was called chronic infectious arthritis, because the clinical and pathologic features of disease resemble those seen in chronic infectious diseases such as tuberculosis. One important host variable is the major histocompatibility locus. Human leukocyte antigen (HLA) genes located within the major histocompatibility complex on chromosome 6p have been found to have a strong association with RA[26].

Rheumatoid arthritis (RA) is the most common systemic inflammatory autoimmune disease, in which joint synovium is primarily affected by a dysregulated immune system. RA is typically associated with serological evidence of systemic autoimmunity, as indicated by the presence of autoantibodies in serum and synovial fluid.

The first autoantibody in RA, rheumatoid factor (RF), was described by Waaler in 1940, and it was later found to be directed to the Fc region of IgG. Autoantigens targeted by a number of autoantibodies, subsequently found in RA, such as cartilage components, stress proteins, enzymes, nuclear proteins and citrullinated proteins, which demonstrates that RA is not characterized by only one autoreactivity to a single autoantigen, but by accumulated autoreactivities in both B and T cells. The spectrum of these self-antigens and immunologically relevant epitopes probably varies during the disease course, and the set of autoantigens in one individual, may differ from that in another. In 1993, Serre et al. identified filaggrin as the target antigen of RA-specific anti-keratin antibodies (AKAs). Subsequently, it has been demonstrated that AKAs and other RA-specific auto-antibodies known as anti-perinuclear factors (APFs), and anti-Sa antibodies, all recognize citrulline-containing peptides/proteins as common antigenic entity, and they are collectively termed as anti- citrullinated protein antibodies (ACPAs). Currently, only RF and ACPA are utilized in clinical practice because of their diagnostic and prognostic values; the latter, in particular, is highly specific for RA.

The association between high titer RF status and a poor prognosis indicates that RF may have a role in the pathogenesis of RA. Furthermore, RF has proven to be the most useful disease marker of RA, as included in the American College of Rheumatology classification criteria for RA.

As with RF, they are associated with more erosive RA. Although ACPAs are also referred to as antiperinuclear factor, antikeratin, antifilaggrin and anticyclic citrullinated

peptide anti-bodies, depending on the antigens used for their detection, citrulline is a common critical constituent of the antigenic determinant of these antibodies, as its absence leads to a lack of recognition by antibodies.

ACPA production is thought to be limited to subjects with certain genetic backgrounds, among which RA-shared epitope (SE) located in the HLA-DRB1 gene is the most dominant genetic factor.

SE, a common region with highly similar sequences among certain HLA-DR class II alleles, is the genetic factor best known to be associated with RA. 46 MHC class II molecules expressing SE can bind and present citrullinated peptides to T cells. Studies utilizing RA patients of North American and several European ancestries, have found that SE alleles are not associated with RA in ACPA negative patients, indicating that these alleles are risk factors for ACPA rather than RA. However, the greatly increased odds ratio for RA susceptibility and radiological progression in individuals with the combination of SE gene carriage and ACPA, than individuals with either of them suggests a synergistic interaction of SE and ACPA[27].

Binding of ACPA to the cell surface increases cellular differentiation to bone-resorbing osteoclasts via autocrine stimulation of TNF production. Induction of osteoclastogenesis by ACPA binding is also highlighted by high levels of markers of bone resorption in the serum of patients with RA, reflecting osteoclast mediated bone resorption[5].

Both erosions and systemic osteoporosis are related to the imbalance between osteoblasts and osteoclasts activity. Some of the cytokines involved in RA physiopathology, as the tumor necrosis factor alpha (TNF-α) and RANKL, are also involved in the pathogenesis of both focal and systemic bone lesions. The balance between the osteoblasts and osteoclasts activity, in course of RA is conditioned not only from distinctive inflammatory factors (TNF-α, IL1, IL6, IL17, IFN-gamma), but also from metabolic factors (IGF1, estradiol, parathyroid hormone, leptin) related to the disease per-se, or influenced by the use of some drugs as glucocorticoids. The fundamental role is played by osteoclasts, both from the synovial and the medullary side[6].

Histopathology of synovial tissue of patients with RA, has revealed an abundance of osteoclasts in bone erosions, but a paucity or even absence of mature osteoblasts, suggesting that certain molecules in the synovium effectively block differentiation of bone- forming osteoblasts[5].

In a population based study in Finland, occurense of HLA-DR4 was 54% in patients with RA and in 30% with control patients[28].

In a study by **Pincus T et al (1994)[29]**, individuals carrying HLA-DR4 and HLA-DR1 alleles in particular, have been shown to posses a higher risk of the disease. It has been suggested that the susceptibility alleles all share a single epitope that is responsible for the predisposition of individuals to RA. (29)

By using the miRNA microarray analysis, **Philippe et al[30],** showed resident cells like fibroblast-like synoviocytes (FLS), play a crucial role in rheumatoid arthritis (RA). They are implicated in the inflammatory response and play a key role in osteoarticular

destruction. Moreover, RA FLS spread RA to unaffected joints. Pathogen-associated molecular patterns and damage-associated molecular patterns have been found to activate RA FLS by interacting with pattern recognition receptors, such as TLR. RA FLS express a large number of TLR, and TLR2 was demonstrated to be involved in RA inflammation. Because microRNA have emerged as important controllers of TLR expression and signaling, the aim of the study was to evaluate their potential involvement in the control of TLR2 expression by RA FLS. It was first showed that TLR2 expression is strongly upregulated in RA FLS, in response to TLR2 ligands. Using a microRNA microarray analysis, they identified that one miRNA in activated RA FLS, miR-19b, which was downregulated and predicted to target Tlr2 mRNA. Downregulation of miR-19b and miR-19a, which belongs to the same cluster, was confirmed by real-time quantitative PCR. Transfection of RA FLS with miR-19a/b mimics, decreased TLR2 protein expression. In parallel, it was found that both IL-6 and matrix metalloproteinase 3 secretion was significantly downregulated in activated FLS, transfected with either mimic. Moreover, using a luciferase assay, it was showed that miR-19a/b directly target TLR2 mRNA. Taken together, the data points toward an important role for miR-19a/b in the regulation of IL-6 and matrix metalloproteinase 3 release by controlling TLR2 expression, as well as provide evidence that miR-19a/b can act as negative regulators of inflammation in humans. These findings provide clear evidence that miR-19a and miR-19b, which are induced by bacterial ligands, can act as negative regulators of inflammation in humans.

MacGregor et al[31], have investigated the association between allotypes of HLA-DR4 and radiological erosion in the peripheral joints of patients with RA. They found that in all patients with RA, DRB1*0401/0404 genotype was associated with a substantially increased risk of being rheumatoid factor positive, and of having subcutaneous nodules and radiological erosion. They suggested that HLA-DRB1*04 might be a marker of severe RA.

CLINICAL FEATURES

- In 1987 classification criteria were developed by the American College of Rheumatology (ACR) to distinguish non-RA from established RA[32].

- Criterion and Definition

1. Morning stiffness

Morning stiffness in and around the joints, lasting at least 1 hour before maximal improvement.

2. Arthritis of 3 or more joint areas

At least 3 joint areas simultaneously have had soft tissue swelling or fluid (not bony overgrowth alone) observed by a physician. The 14 possible areas are right or left PIP, MCP, wrist, elbow, knee, ankle, and MTP joints.

3. Arthritis of hand joints

At least 1 area swollen (as defined above) in a wrist, MCP, or PIP joint

4. Symmetric arthritis

Simultaneous involvement of the same joint areas (as defined in 2) on both sides of the body (bilateral involvement of PIPs, MCPs, or MTPs is acceptable without absolute symmetry).

5. Rheumatoid nodules

Subcutaneous nodules, over bony prominences, or extensor surfaces, or in juxta-articular regions, observed by a physician.

6. Serum rheumatoid factor

Demonstration of abnormal amounts of serum rheumatoid factor by any method, for which the result has been positive in <5% of normal control subjects

7. Radiographic changes

Radiographic changes typical of RA, on postero-anterior hand and wrist radiographs, which must include erosions or unequivocal bony decalcification, localised in or most marked adjacent to the involved joints (osteoarthritis changes alone do not qualify)

*For classification purposes, a patient shall be said to have RA if he/she has satisfied at least 4 of these 7 criteria. Criteria 1 through 4 must have been present for at least 6 weeks. Patients with 2 clinical diagnoses are not excluded. Designation as classic, definite, or probable RA is not to be made.

Previously, Rheumatoid Arthritis (RA) was defined by the presence of four of seven criteria set by the American College of Rheumatology (ACR). In 2010, the ACR

collaborated with the European League against Rheumatism (EULAR) and developed a new classification criteria which focuses on patients with synovitis for less than 6 weeks of whom symptoms are not better explained by another diagnosis. The new criteria set are thought to be more sensitive in identifying patients in the early stages of RA prior to developing erosive disease. Therefore, allowing for the early initiation of DMARD therapy. Scoring is based on 4 categories: 0-5 points for number and site of involved joints, 0-3 points for serologic abnormality, 0-1 points for elevated acute phase response and 0-1 points for the duration of symptoms. A total score of 6 or more increases the possibility of RA[33].

The clinical history of a patient with RA, generally includes pain and symmetric swelling involving multiple joints. Any joint may be affected, but those most commonly involved are the metacarpophalangeal joints of the hand, proximal interphalangeal joints of the hand, metatarsophalangeal joints, wrists, knees, elbows, and, less commonly, shoulder, hip, and distal interphalangeal joints[29].

RF may represent a marker of the earliest phases of the pathogenetic process of RA, that may be detectable before the appearance of other features that permit a clinical diagnosis. A study by **Del Puente A et al (1988)[34]**, with 2,712 Pima Indians of Arizona, 20 years of age or older, initially without RA, was observed for up to 19 years with biennial examinations. It concluded that the presence of RF, in subjects without RA, is a risk factor for the development of RA, and that this risk is related to the RF titer.

Another study by **Sune F Nielsen et al, (2012)[35]** included white Danish individuals from the general population aged 20-100 years without rheumatoid arthritis at

study entry. Individuals in the general population, with elevated rheumatoid factor, have up to 26-fold greater long term risk of rheumatoid arthritis, and up to 32% 10 year absolute risk of rheumatoid arthritis. These novel findings may lead to revision of guidelines for early referral to a rheumatologist and early arthritis clinics based on rheumatoid factor testing.

Anti- CCP is highly specific for diagnosing rheumatoid arthritis**. Nishimura et al, (2007)**[36], concluded that the specificity of anti-CCP for the diagnosis of rheumatoid arthritis was greater than that of RF. Anti-CCP may also be a better predictor of erosive disease.

Study by **Panchagnul R et al (2006)**[37], suggested that anti-CCP is a useful and highly specific, but not absolutely specific or sensitive test to detect RA, and supplements rheumatoid factor in the presence of a strong clinical suspicion.

However ,it was proposed by **Avouac et al (2006)**[38], that the sensitivity of the second generation of anti-CCP is close to that of rheumatoid factor, with a higher specificity, for distinguishing RA from other rheumatic diseases. Moreover, anti-CCP antibodies appear to be highly predictive of the future development of RA in both healthy subjects and patients with undifferentiated arthritis.

TEMPOROMANDIBULAR JOINT

The area where the mandible articulates with the cranium, the TMJ, is one of the most complex joints in the body. It provides for hinging movement in one plane and therefore can be considered a ginglymoid joint. However, at the same time, it also provides for gliding movements, which classifies it as an arthrodial joint. Thus it has been technically considered a ginglymoarthrodial joint. The TMJ is formed by the mandibular condyle fitting into the mandibular fossa of the temporal bone. Separating these two bones from direct articulation is the articular disc. In the normal joint, the articular surface of the condyle is located on the intermediate zone of the disc, bordered by the thicker anterior and posterior regions. The condyle is the portion of the mandible that articulates with the cranium around which movement occurs.The total mediolateral length of the condyle is between 18 and 23mm, and the anteroposterior width is between 8 and 10 mm. The actual articulating surface of the condyle extends both anteriorly and posteriorly to the most superior aspect of the condyle. The posterior articulating surface is greater than the anterior surface. The articulating surface of the condyle is quite convex anteroposteriorly and only slightly convex mediolaterally. The mandibular condyle articulates at the base of the cranium with the squamous portion of the temporal bone. This portion of the temporal bone is made up of a concave mandibular fossa, in which the condyle is situated and which has also been called the articular or glenoid fossa. Posterior to the mandibular fossa is the squamotympanic fissure. Immediately anterior to the fossa is a convex bony prominence called the articular eminence. The degree of convexity of the articular eminence is highly variable, but important, because the steepness of this surface dictates the pathway of the condyle when the mandible is positioned anteriorly[39].

Although rheumatoid arthritis is more commonly associated with the joints of the hands, it also may occur in the TMJs and is then almost always bilateral. A history of multiple joint complaints is a significant diagnostic finding. About 50% of patients with rheumatoid arthritis will present with TMJ complaints.

About 80% of rheumatoid patients are seropositive for rheumatoid factor. Although not conclusive, this test is helpful in identifying rheumatoid arthritis. In one radiographic study, two thirds of the patients with rheumatoid arthritis demonstrated erosive changes in the TMJs[39].

Clinical characteristics of rheumatoid arthritis includes pain/tenderness at the TMJ region, morning stiffness, joint sounds such as crepitus or clicking, limitation of mouth opening and swelling over joint area[40].

Yamakawa M et al (2002)[41] conducted a study on 142 women with rheumatoid arthritis (40 to 69 years) and 143 women of similar age without RA to estimate the prevalence of TMJ disorder in RA subjects. In the RA group, TMJ tenderness was elicited in 9.2%, clicking in 12.7%, and crepitus in 35.9%, representing a significant excess occurrence of crepitus. They concluded the prevalence of TMJ disorders in RA patients to be 67.6%.

Yi-Chun Lin et al (2007)[42] recruited 56 adult RA patients and were found to have a very high prevalence of TMD (92.9%). They concluded that TMD is a frequent manifestation of RA. Approximately half of the cases of TMJ involvement in RA developed profound symptoms or joint abnormalities.

Radiological investigations are of paramount importance in the diagnostic assessment of a patient with TMD. The American Academy of Oral and Maxillofacial Radiology (AAOMR) has established the rationale for image selection for diagnosis, treatment planning and follow up of a patient with conditions affecting the TMJ. Conventional radiographic TMJ projections like transpharyngeal, transcranial, panoramic radiograph, conventional tomographic sections of TMJ may be adequate in a number of clinical situations. But there are bony alterations that occur in these disorders like erosions, osteophytes, pneumatisation of articular eminence that are difficult to be detected in conventional radiographs due to overlapping of the anatomic structures. This warrants the use of advanced imaging modalities like Magnetic Resonance Imaging, arthrography, conventional Computed Tomography and Cone Beam Computed Tomography[43].

Celiker R et al (1995)[44] included twenty patients in a study, ten were evaluated with computed tomography and ten with magnetic resonance imaging. Among the 20 patients, 45% had TMJ involvement detected by imaging techniques. The most frequent pathological signs were osteophyte formation, erosion of the mandibular condyle and decreased joint space (40%). They concluded that TMJ involvement may be detected even in asymptomatic patients with RA and there is a positive correlation between the severity of disease and involvement of TMJ. The prevalence of TMJ involvement in RA may be as high as 45%, the majority of respondents estimated that only as many as 25% of their patients with RA had TMJ involvement.

Goran W.Gynther et al (1991)[45] studied 20 patients with generalized osteoarthritis and 21 patients with rheumatoid arthritis and concluded that no

radiographic criterion pathognomonic for generalized osteoarthritis or rheumatoid arthritis, However, osteophytes, flattening of condyle or reduced joint space is found to be more in generalized osteoarthritis and erosions in condyle more in rheumatoid arthritis.

A study by **Dahlstrom L et al (1996)**[46] was conducted to assess the reliability and validity of panoramic radiography for TMJ evaluation in comparison with tomography. TMJ was assessed for 50 TMD patients and 20 non-TMD subjects. They concluded that evaluation of bony changes on the condyles has acceptable reliability and specificity, but low sensitivity, whereas the temporal component has low reliability and accuracy. If bony abnormality is suspected in the TMJ, tomography may be indicated than panoramic radiography.

As conventional plain radiographs of various TMJ projections depict only mineralized structures of the TMJ. These are, however, plagued by the numerous superimpositions of the adjacent structures which make visualization cumbersome[43].

Cone Beam Computed Tomography (CBCT)

It is a recent innovation in field of technology that has achieved the rapid acceptance in general, particularly in dentistry despite its current relatively high price when compared with alternative imaging methodologies. Craniofacial CBCTs were designed to counteract some of the limitations of the conventional CT scanning devices[47].

The object to be evaluated is captured as the radiation source falls onto a two-dimensional detector. This simple difference allows a single rotation of the radiation source to capture an entire region of interest, as compared to conventional CT devices where multiple slices are stacked to obtain a complete image[48]. The cone beam also produces a more focused beam of x-ray and significantly less scatter radiation compared to the conventional fan-shaped CT devices, and this considerably increases the X-ray utilization and reduces the ability of X-ray tube required for volumetric scanning[49].

It has been reported that the total radiation dose is approximately 20% of conventional CTs and equivalent to a full mouth periapical radiographic exposure[50].

Presently, the available CBCT equipment differs in size, possible settings, area of image capture (field of view), and clinical usage.

CBCT has application in several diagnostic areas, such as implant treatment, oral surgery, endodontic treatment, and temporomandibular joint imaging. The great advantage of this technology is that offers 3-dimensional (3D) imaging of dental structures and provides clear images of highly contrasted structures, such as bone. In

comparison to the conventional computed tomography, CBCT technology in clinical practice has significant advantages such as minimization of the dose of radiation to the patient, accuracy of image, rapid scanning time, lesser image artifacts, chair-side image display, high spatial resolution and real-time analysis[51, 52].

PRINCIPLE OF CONE BEAM COMPUTED TOMOGRAPHY

All CT scanners consist of an x-ray source and detector mounted on a rotating gantry. During rotation of the gantry, the receptor detects x rays attenuated by the patient. These recordings constitute raw data that is reconstructed by a computer algorithm to generate cross sectional images, whose component picture element (pixel) values correspond to linear attenuation coefficients. CT can be divided into 2 categories on the basis of acquisition of x ray beam geometry, namely fan beam and cone beam.

Cone beam scanners use a 2 dimensional digital array providing an area detector, unlike linear detector as CT does. This is combined with a three dimensional (3D) x-ray beam with circular collimation so that the resultant beam is in the shape of a cone, hence the name cone beam because the exposure incorporates the entire region of interest (ROI), only one rotational scan of the gantry is necessary to acquire enough data for reconstruction. Cone beam geometry has inherent quickness in volumetric data acquisition and therefore the potential for significant cost savings as compared with CT, CBCT produces an entire volumetric dataset from which the voxels are extracted. Voxel

dimensions are dependent on the pixel size on the area detector. Therefore CBCT units in general provide voxel resolutions that are isotropic- equal in all three dimensions[53].

CBCT technology has a substantial impact on the maxillofacial imaging. It has been applied to diagnosis in almost all the areas of dentistry and now its role is also expanding into treatment fields such as temporomandibular joint, Implant site assessment, Orthodontics and Three Dimensional Cephalometrics, Conditions of the maxillofacial complex, Odontogenic Cysts and Tumors[54].

Hussain et al.(2008)[55] in their systematic review evaluated the ability of different diagnostic imaging techniques for diagnosing the presence of erosions and osteophytes in the temporomandibular joint (TMJ) and mentioned that axially corrected sagittal tomography is currently the imaging modality of choice for diagnosing erosions and osteophytes in the TMJ. They put forward that CT does not seem to add any significant information to what is obtained from axially corrected sagittal tomography. CBCT might prove to be a cost and radiation dose-effective alternative to axially corrected sagittal tomography. They concluded that combining different radiographic techniques is likely to be more accurate in diagnosing erosions and osteophytes in the TMJ than using a single imaging modality[55].

A study by **KE Alexiou et al (2009)[56]** on CBCT, to evaluate and correlate with age the severity of TMJ osteoarthritic changes using CBCT, and concluded that older patients are expected to have more frequent and severe progressive degenerative bony changes, than patients in younger age groups.

Alkhader M, et al[57] performed examination of 106 TMJs from 55 patients with temporomandibular disorder by CBCT and MRI. Their results showed that MRI was better at detecting changes in the size of the TMJ, such as deformities, than it was at detecting changes in shape, for example flattening, osteophyte formation or erosion. This fact could be due primarily to the limited spatial resolution of MRI; the slice thickness of MRI is 3 mm or more for clinical use, which may be too thick to detect subtle osseous changes. Other problems include the presence of fibrous tissues inside the TMJ and the attachment of the lateral pterygoid muscle in close proximity to the articular surface of the condyle, which can be interpreted as either an osseous abnormality or as a disc, and may result in false-positive or false-negative results. Thus, they concluded that the value of MRI for the detection of TMJ osseous abnormalities is considered to be limited.

JB Ludlow et al (2003)[58] conducted a study on effective dose measurements for two extra oral direct digital imaging devices, cone beam CT unit and panoramic unit and concluded that CBCT examinations resulted in doses that were 3 – 7 times the panoramic doses observed in this study.

Hintze et al(2007)[59] compared the diagnostic accuracy of cone beam CT images with conventional tomographic images for the detection of morphological temporomandibular joint (TMJ) changes and resulted no significant differences in diagnostic accuracy between the two techniques. They concluded that there is no evidence, that conventional or CBCT is inferior to CT, the choice of imaging technique must depend on the available equipment.

MATERIALS AND METHODS

The study was conducted among the patients referred from department of rheumatology, Rajiv Gandhi Government General hospital, Chennai to the Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital with TMJ complaints.

The study protocol was approved by the Institutional Ethical Committee.

DURATION OF THE STUDY: From July 2015- July 2016

SAMPLE DESIGN:

Totally 50 cases were included under the study, of either sex, who were diagnosed for TMJ involvement with RA based on history, clinical findings and laboratory values. The patients were selected who satisfied the following inclusion and exclusion criteria.

Inclusion criteria:

1- Patient diagnosed with rheumatoid arthritis and having TMJ involvement.
2- Between the age of 25 to 60 years, both gender
3- Willing to participate in study.

Exclusion criteria:

1- Patients with myogenous cause of pain.
2- Patients with history of trauma or surgery at TMJ or with ankylosis .
3- Pregnant women
4- Not willing to participate in study

METHODOLOGY:

Study subjects of 50 patients were included in the study, based on inclusion and exclusion criteria, and those who satisfied the clinical criteria of rheumatoid arthritis with TMJ involvement. All those participants were explained about the design of the study, the need for thorough clinical examination and CBCT as a part of the study. Patients who gave a signed informed consent on an institutionally approved document were included in the study. Patient also subjected to routine blood investigations, to rule out any systemic illness, in our institution before the start of the study.

Patients were diagnosed with rheumatoid arthritis based on laboratory values for rheumatoid factor antibody and anti-citrullinated protein antibodies. A detailed case history of the patients, with emphasis on symptoms of rheumatoid arthritis like multiple joint pain, along with TMJ symptoms such as pain/tenderness at the TMJ region, joint sounds such as crepitus and limitation of mouth opening, was recorded. Thorough clinical examination was recorded on a structured proforma designed for the study (Figure 2). A clinical diagnosis of TMJ RA was made and CBCT for each patient involving 100 TMJ was taken in Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College and Hospital, Chennai–600 003.

Armamentarium (Figure 1):

Examination of the patient

1) Electrically operated dental chair
2) Patient's apron
3) Disposable mouth mask
4) A pair of disposable latex examination gloves
5) Stainless steel kidney trays
6) Mouth mirror
7) Stainless steel probe
8) Tweezer
9) Divider and Metallic scale

CS 9300 Select (Carestream Health, Inc.) cone beam 3D extraoral imaging system was used. The CBCT machine had a scanning time of 12-28 seconds (+/- 10%), voxel size 90μm to 180μm, field of view 5x5cm, 8x8cm, 10x5cm, 10x10cm and fitted with a TFT sensor. Exposure parameters for the patients varied from, tube voltage 60 - 90 kV, tube current 2 - 15 mA, with a scan time of 12- 28 seconds. Routine radiation safety procedures were followed. Patients were positioned in standing position while taking the scan. The total image acquisition time was less than 2 minutes. Radiation exposure for a Single CBCT scan was in the range 0.02 to 0.08 mSv. The radiographic exposure for patients was well below the maximum permissible dose of 2.4 mSv as per the NCRP guidelines (Figure 3).

The 3 D volumetric image data and the various sections were viewed in the

Dental Imaging Software 6, 13, 1, 8(Copyright Carestream Health, Inc.,2013) on Hewlett- Packard HP Z220 CMT Workstation running on Windows 7 Professional Operating System (Copyright © 2009 Microsoft Corporation). The DICOM data was analyzed on secondary reconstructed orthogonal slices, reformatted OPG and reconstructed 3 dimensional images. Cross sectional CBCT images were evaluated to assess the osseous changes at temporomandibular joint in rheumatoid arthritis patients. Assessment was done for the condyle of the mandible, glenoid fossa and joint space.

1. To interpret the CBCT axial, coronal, sagittal and 3 dimensional images of osseous changes of condyle of the mandible in rheumatoid arthritis patients in relation to the morphological characteristics like

- **Erosion** - defined as area of decreased density of the cortical bone and adjacent subcortical bone of the condyle.
- **Flattening** - defined as a flat bony contour deviating from convex form.
- **Osteophyte**- defined as marginal bony outgrowth of the condyle.
- **Sclerosis**- defined as an area of increased density of cortical bone extending into the bone marrow. (56)
- **Subchondral cyst** - defined as a space of ≥1mm in diameter and connecting with the cartilage loss regions (60)

2. To interpret the CBCT axial, coronal, sagittal and 3 dimensional images of involved glenoid fossa in rheumatoid arthritis patients in relation to the morphological characteristics like erosion, flattening and sclerosis

3. To interpret the CBCT axial, coronal, sagittal and 3 dimensional images of involved joint space in rheumatoid arthritis patients in relation to the morphological characteristics like normal or reduced joint space.

FOR CHONDYLAR OSSEOUS CHANGES

For analysing, a four point rating scale (0 to 3) was used to define the severity of erosion in the condylar head as follow (Figure 4):

0- Absence of erosion

1- Mild erosion- when decreased density is observed only in the cortical bone.

2- Moderate erosion- when decreased density is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone

3- Severe erosion- when decreased density is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

A four point rating scale (0 to 3) was used to define the severity of flattening in the condylar head as follow (Figure 5):

0- Absence of flattening

1- Mild flattening- when flattening is observed only in the cortical bone.

2- Moderate flattening- when flattening is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone.

3- Severe flattening- when flattening is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

A four point rating scale (0 to 3) was used to define the severity of sclerosis in the condylar head as follow (Figure 6):

0- Absence of sclerosis

1- Mild sclerosis- when sclerosis is observed only in the cortical bone.

2- Moderate sclerosis- when sclerosis is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone

3- Severe sclerosis- when sclerosis is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

A four point rating scale (0 to 3) was used to define the severity of osteophyte in the condylar head as follow (Figure 7):

0- Absence of osteophyte

1- Mild, when marginal bony outgrowth on the condyle was less than 0.5 mm

2- Moderate, when marginal bony outgrowth on the condyle was 0.5- 1 mm

3- Severe, when marginal bony outgrowth on the condyle was more than 1 mm.

A three point rating scale (0 to 2) was used to define the severity of erosion in the condylar head as follow (Figure 8):

0- Absence of subchondral cyst

1- Presence of single subchondral cyst.

2- Presence of multiple subchondral cysts

FOR GLENOID FOSSA OSSEOUS CHANGES

A four point rating scale (0 to 3) was used to define the severity of erosion in the glenoid fossa as follow:

0- Absence of erosion

1- Mild erosion- when decreased density is observed only in the cortical bone.

2- Moderate erosion- when decreased density is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone

3- Severe erosion- when decreased density is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

A four point rating scale (0 to 3) was used to define the severity of flattening in the glenoid fossa as follow (Figure 10):

0- Absence of flattening

1- Mild flattening- when flattening is observed only in the cortical bone.

2- Moderate flattening- when flattening is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone.

3- Severe flattening- when flattening is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

A four point rating scale (0 to 3) was used to define the severity of sclerosis in the glenoid fossa as follow:

0- Absence of sclerosis

1- Mild sclerosis- when sclerosis is observed only in the cortical bone.

2- Moderate sclerosis- when sclerosis is observed in the cortical bone and extends to the upper layers of the adjacent subcortical bone

3- Severe sclerosis- when sclerosis is observed in the cortical bone and extends below the upper layers of the adjacent subcortical bone.

JOINT SPACE

The measurement between the condyle and mandibular fossa was performed on lateral slices at the subjectively closest distance between condyle and mandibular fossa using measurement tool in the CBCT software (Figure 9).

1- Normal – when the distance between the condylar head and mandibular fossa was between 1.5 mm and 4 mm.

2- Reduced- when the distance between the condylar head and mandibular fossa was less than 1.5 mm.

STATISTICAL ANALYSIS-

The statistical analysis was done using the computer software program SPSS version 18.01. All data are expressed as mean rank scores. Descriptive statistics for various clinical and radiographic parameters was present as percentage for qualitative data. Arithmetic mean and standard deviation were estimated for quantitative data. The percentage of distribution of age groups and sex was also calculated.

Comparison of radiographic features based on clinical finding and Statistical significance was determined using the Mann-Whitney test.

Comparison of frequency of radiographic parameters like erosion, flattening, sclerosis, subchondral cyst and osteophyte in different CBCT sections like axial, coronal, sagittal and 3D reconstructed view was performed by Pearson chi square test.

The smaller the p value, the more significant the result was said to be. All p values are two tailed, and confidence intervals were calculated at the 95% level. In the present study, $p < 0.05$ was considered as the level of significance.

PHOTOGRAPHS

FIGURE 1: ARMAMENTARIUM

FIGURE 2: CLINICAL EXAMINATION OF TMJ

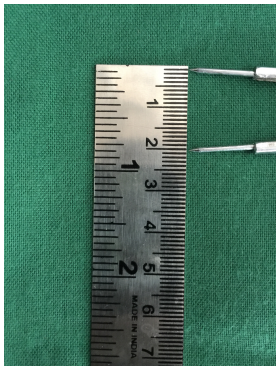


FIGURE 3A : CBCT MACHINE

FIGURE 3B : EXPOSURE CRITERIA

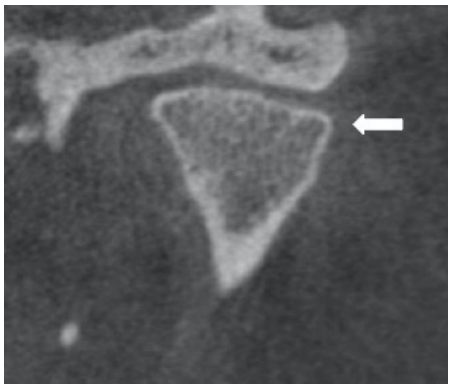
FIGURE 4 : CBCT REVEALING CONDYLAR EROSION

Mild erosion

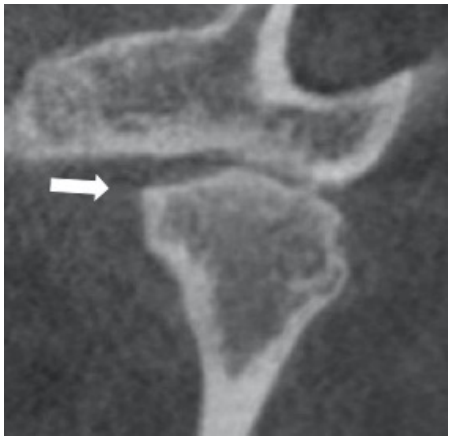
Moderate erosion

Severe erosion

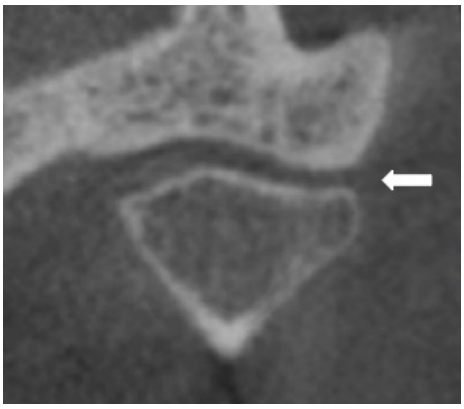
FIGURE 5 : CBCT REVEALING CONDYLAR FLATTENING



Mild flattening



Moderate flattening



Severe flattening

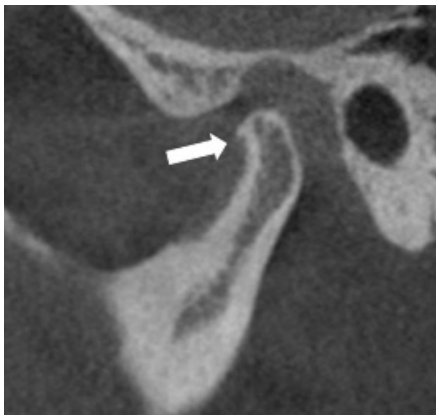
FIGURE 6 : CBCT REVEALING CONDYLAR SCLEROSIS

Mild sclerosis

Moderate sclerosis

Severe sclerosis

FIGURE 7 : CBCT REVEALING CONDYLAR OSTEOPHYTE

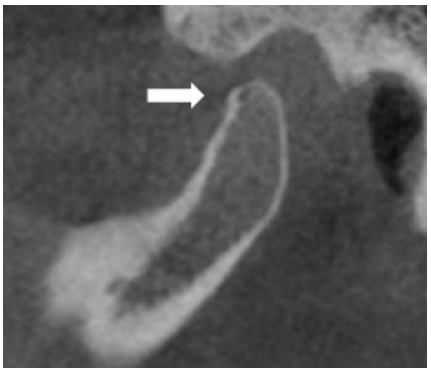


Mild osteophyte

Moderate osteophyte

Severe osteophyte

FIGURE 8 : CBCT REVEALING SUBCHONDRAL CYST

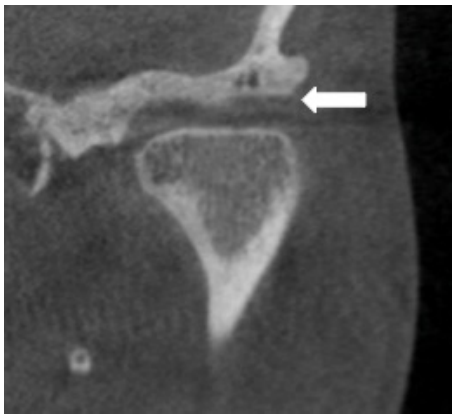


Single subchondral cyst

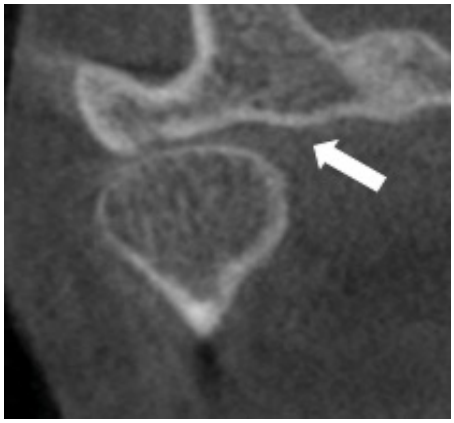
Multiple subchondral cysts

FIGURE 9 : CBCT REVEALING REDUCED JOINT SPACE

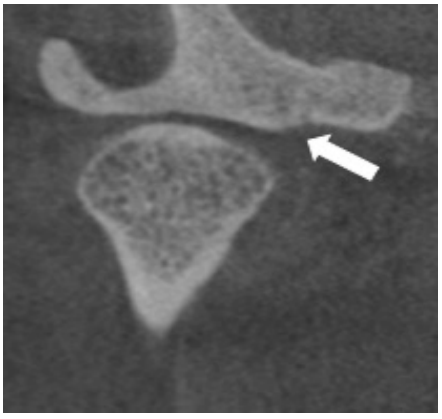
FIGURE 10 : CBCT REVEALING GLENOID FOSSA FLATTENING



Mild flattening



Moderate flattening



Severe flattening

CHART 1: Sex distribution

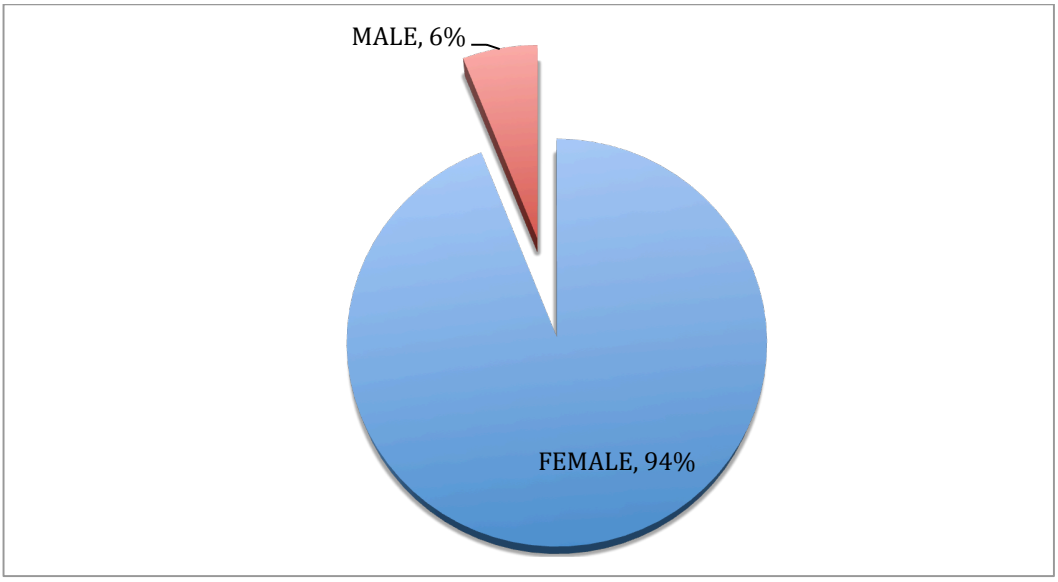


CHART 2- Distribution of severity of erosion In left condyles

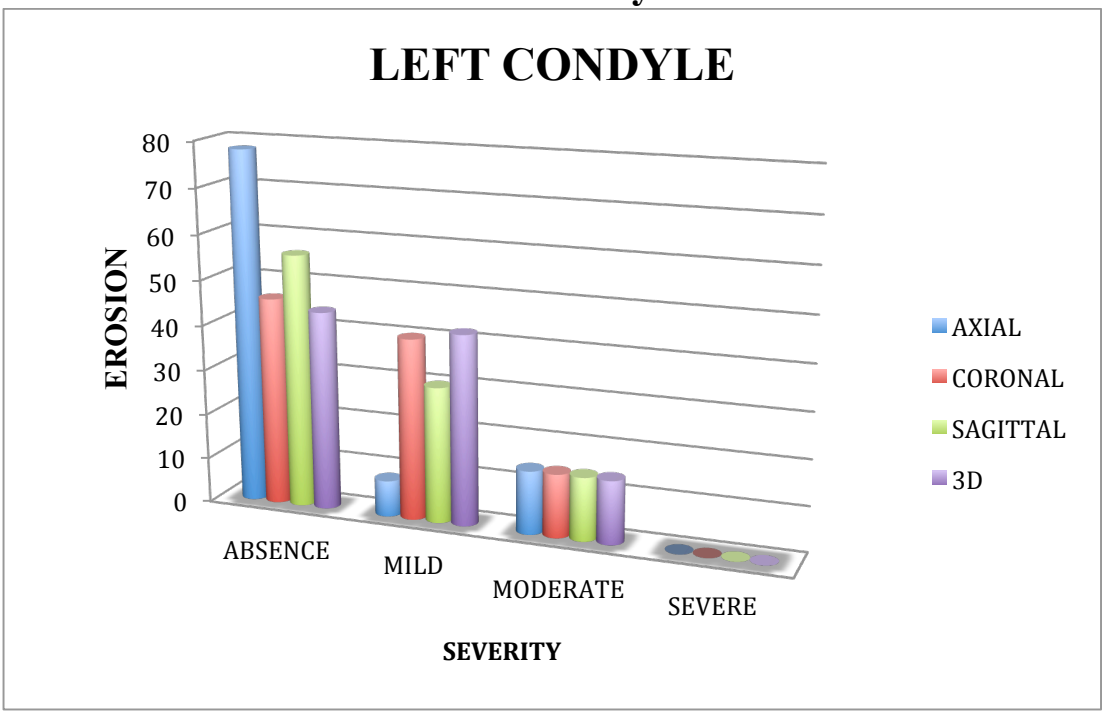


CHART 3- Distribution of severity of sclerosis In left condyles

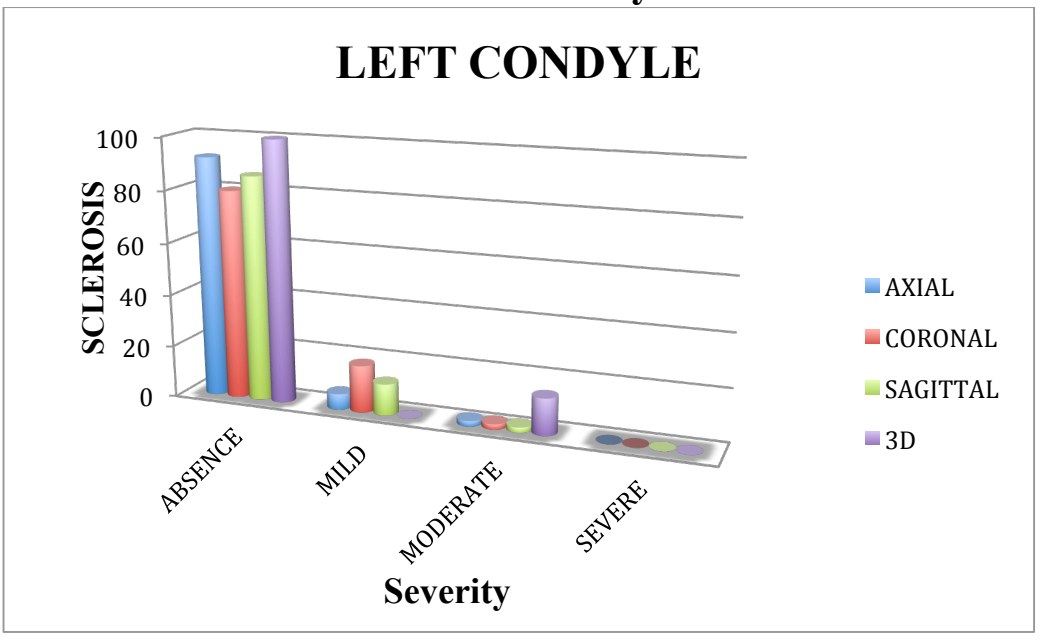


CHART 4- Distribution of severity of subchondral in left condyles

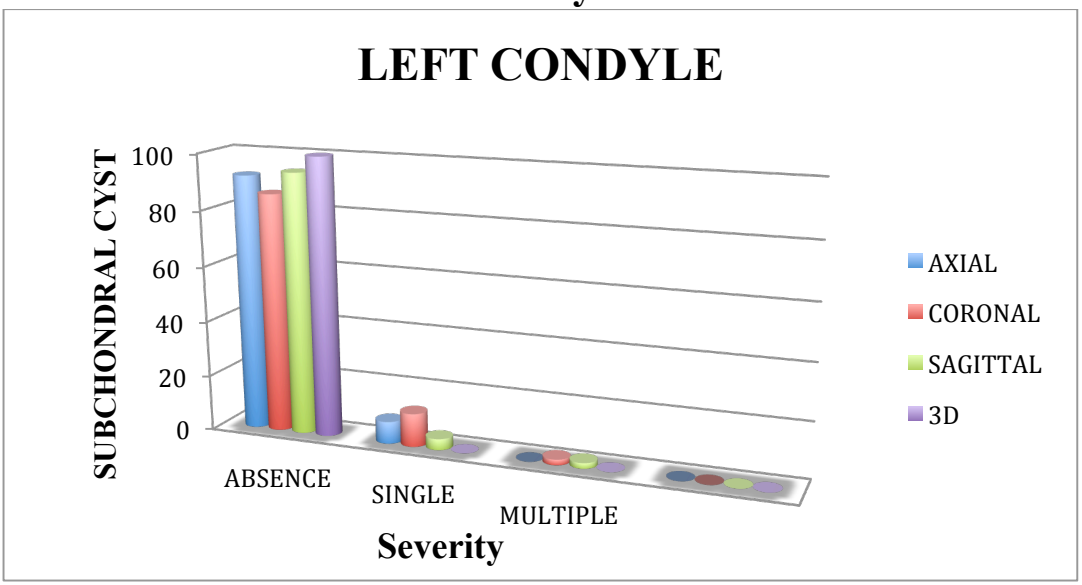


CHART 5- Distribution of severity of osteophyte in left condyles

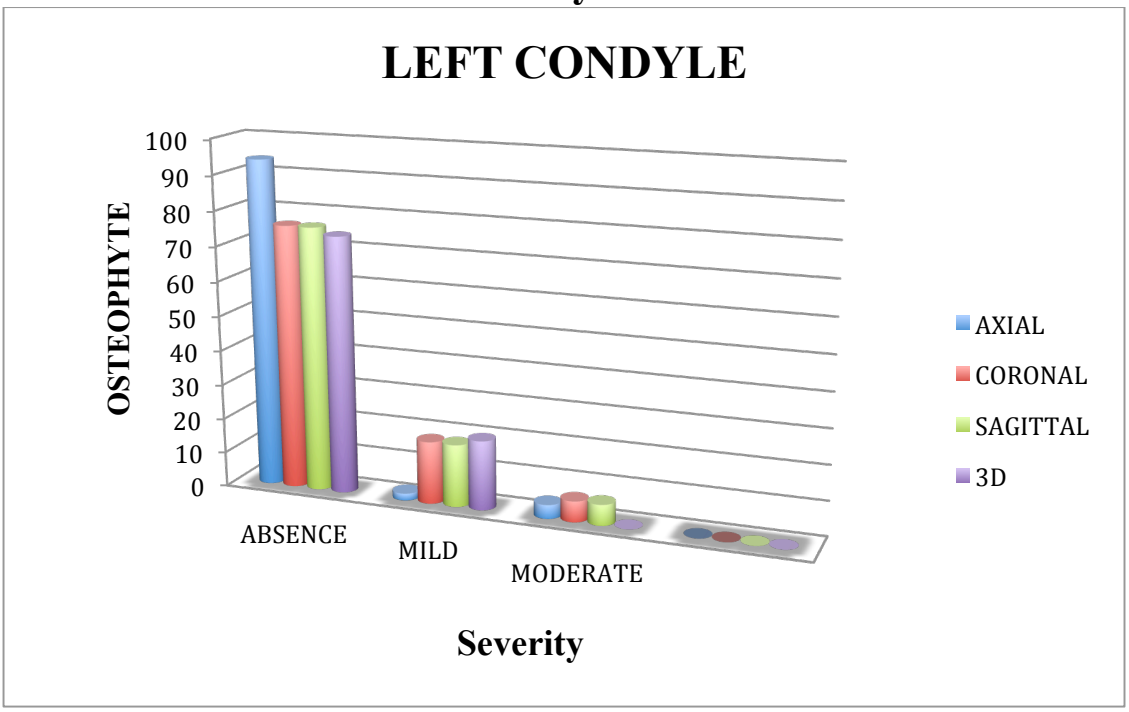


CHART 6- Distribution of severity of erosion in right condyles

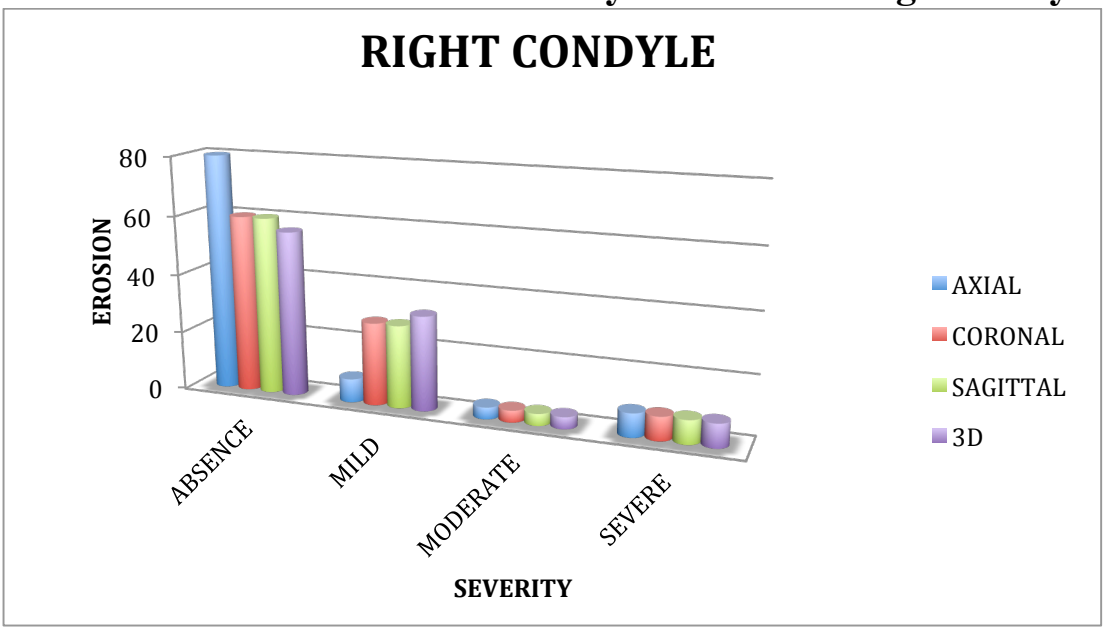


CHART 7- Distribution of severity of sclerosis in right condyles

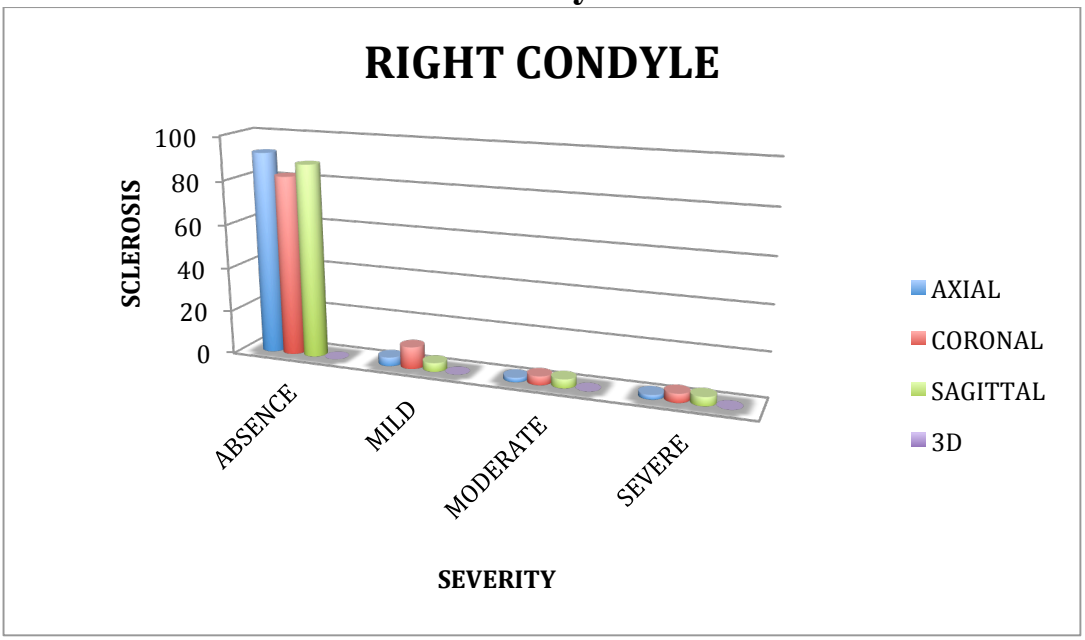


CHART 8- Distribution of severity of subchondral cyst in right condyles

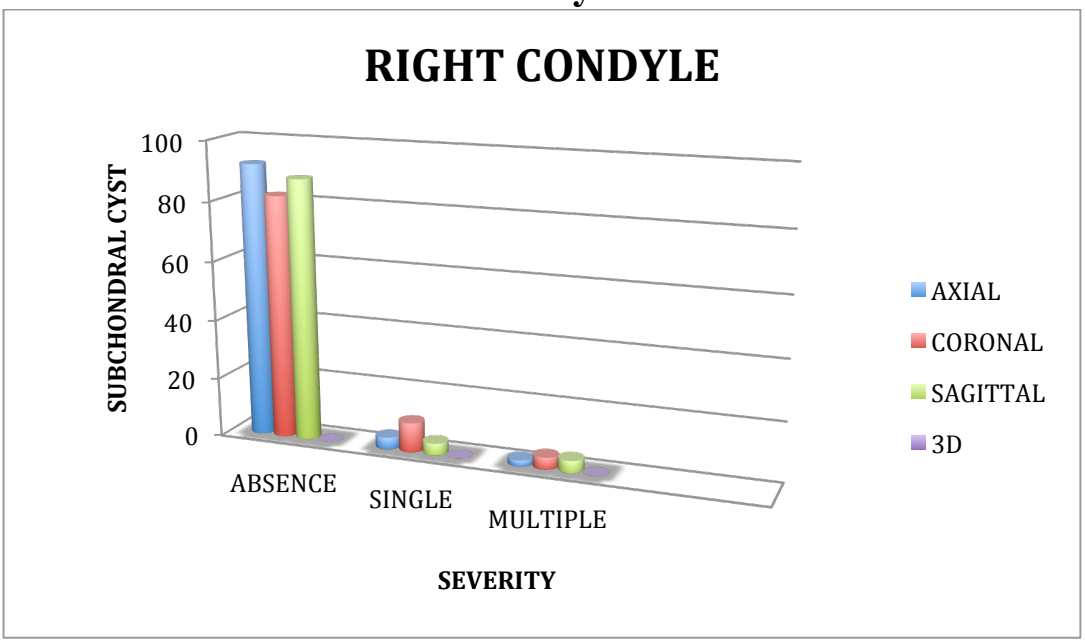


CHART 9- Distribution of severity of osteophyte in right condyles

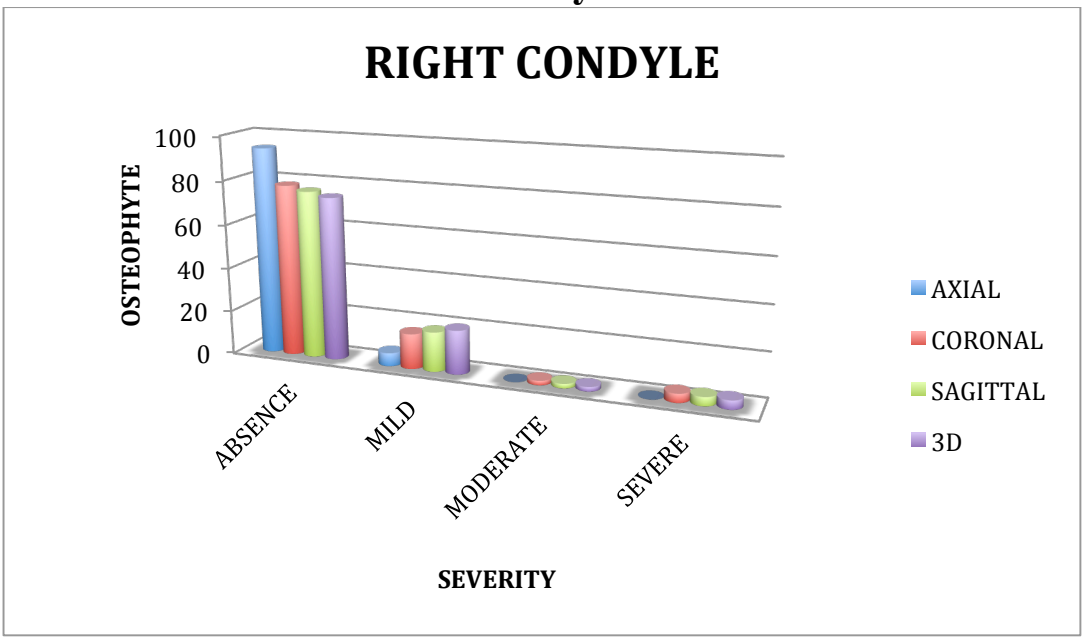


CHART 10- Distribution of severity of erosion in left glenoid fossa

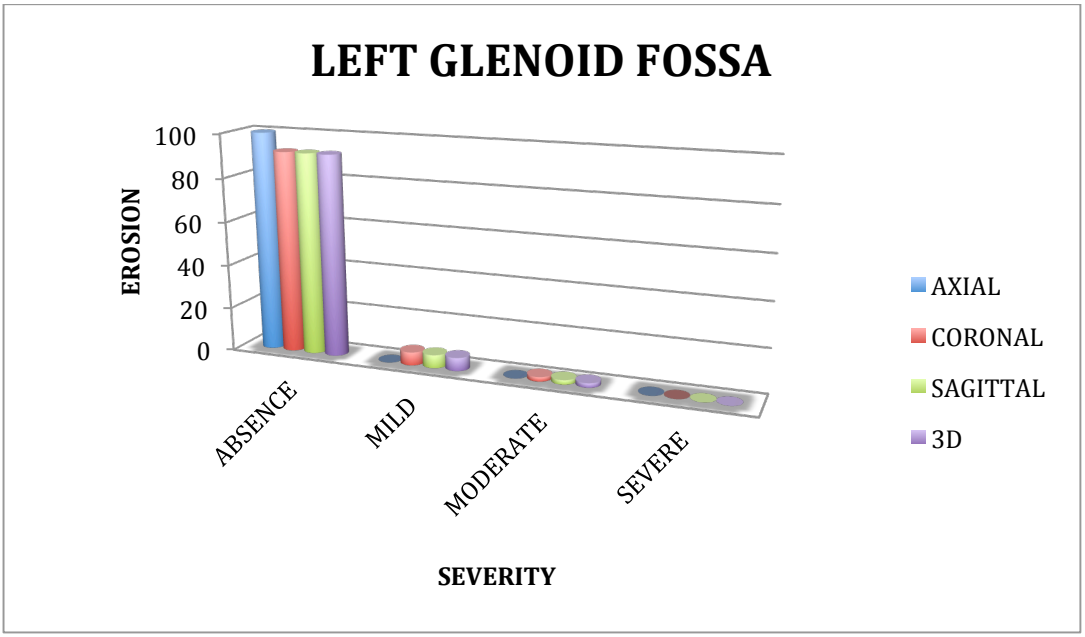


CHART 11- Distribution of severity of flattening in left glenoid fossa

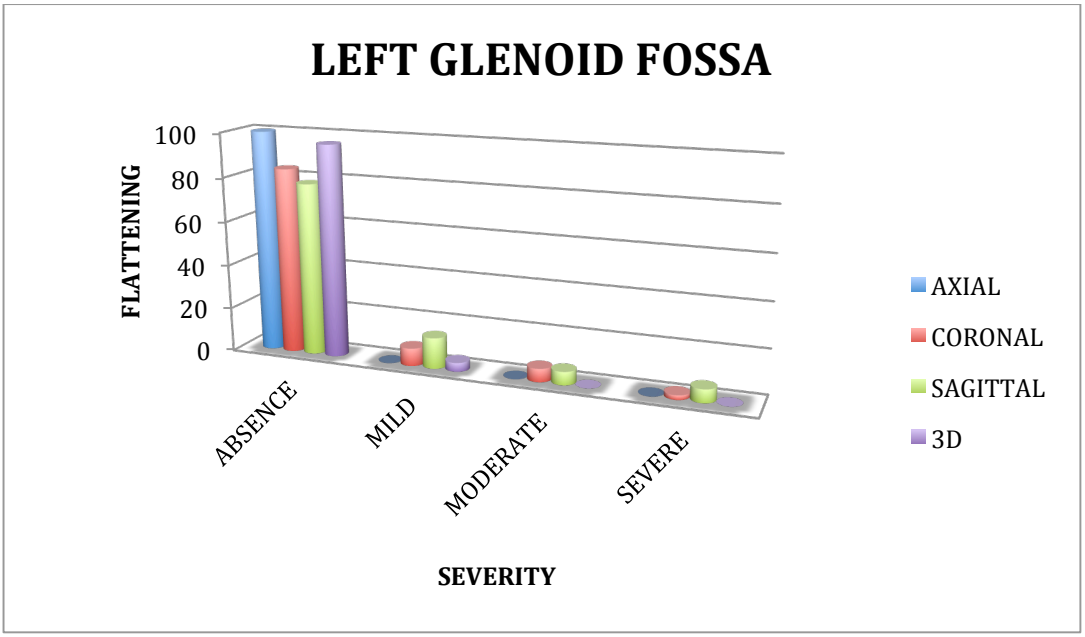


CHART 12- Distribution of severity of sclerosis in left glenoid fossa

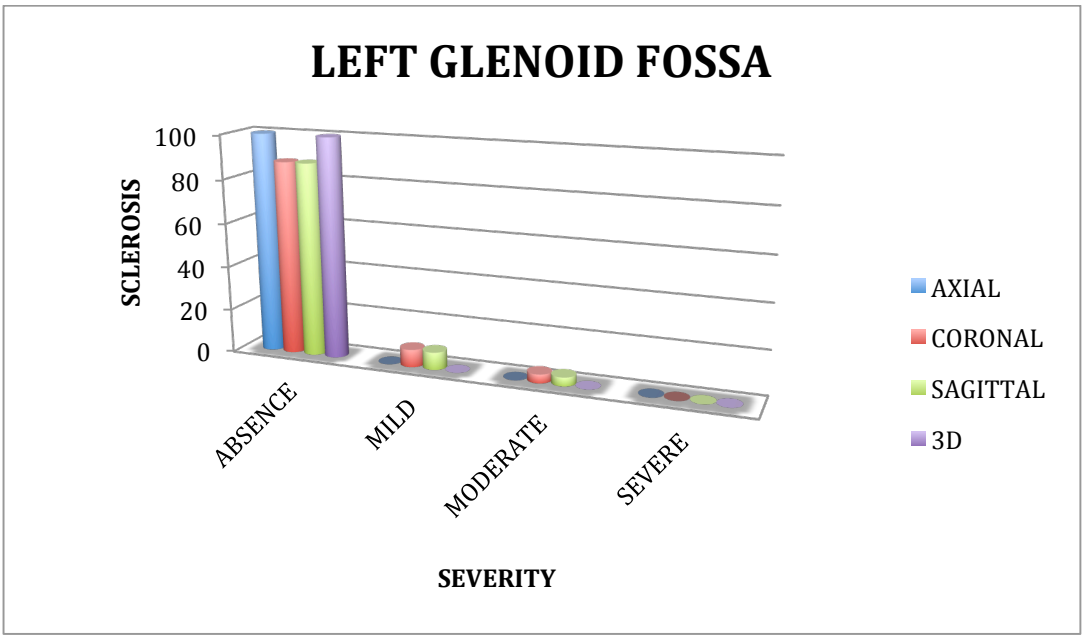


CHART 13- Distribution of severity of erosion in right glenoid fossa

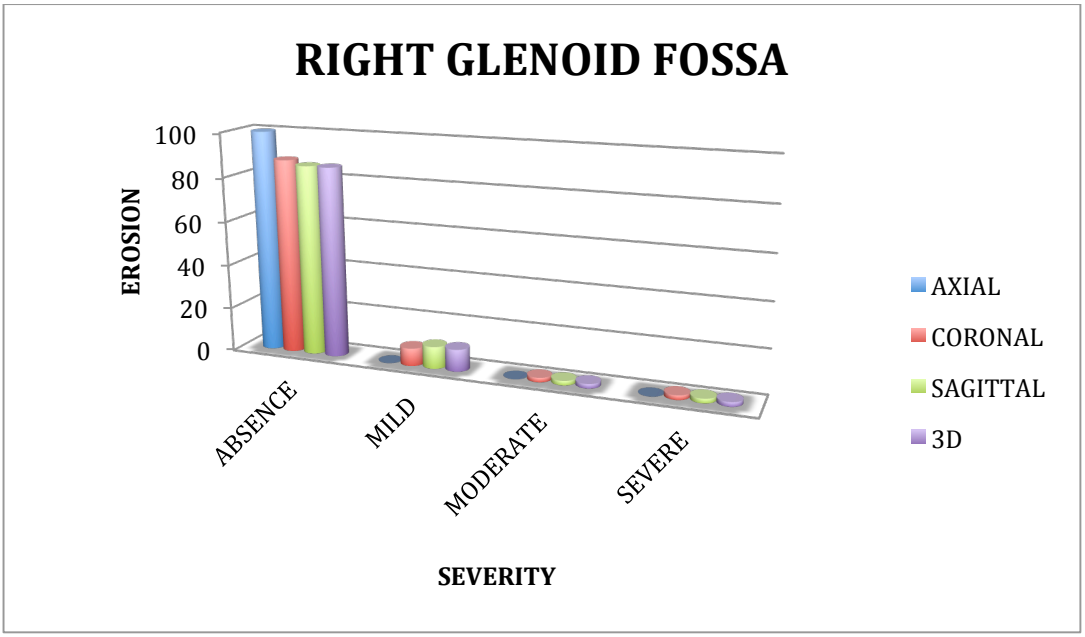


CHART 14- Distribution of severity of flattening in right glenoid fossa

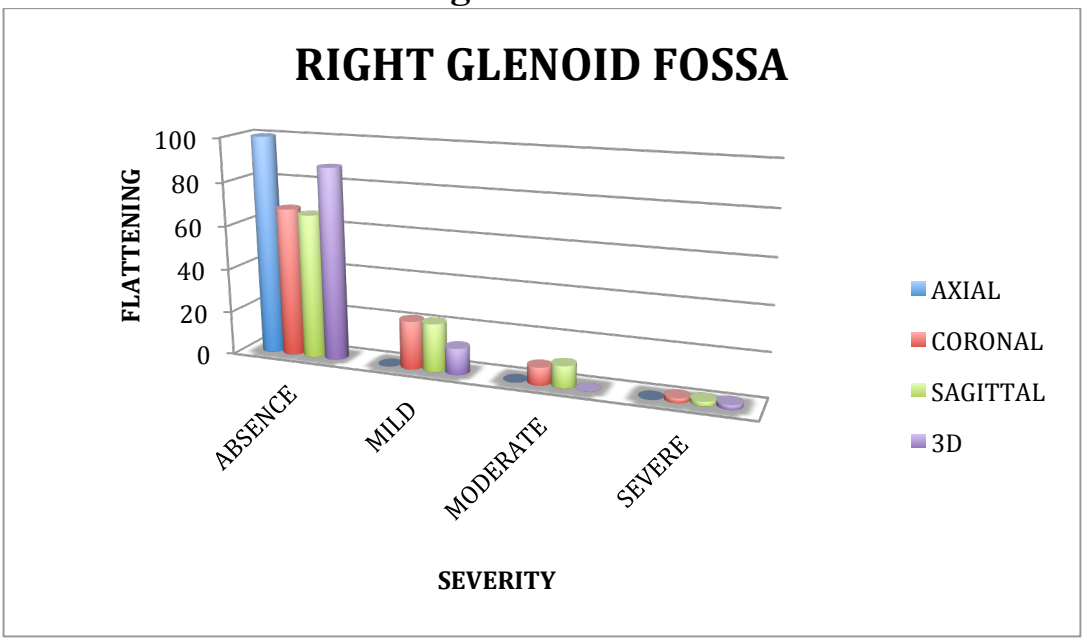
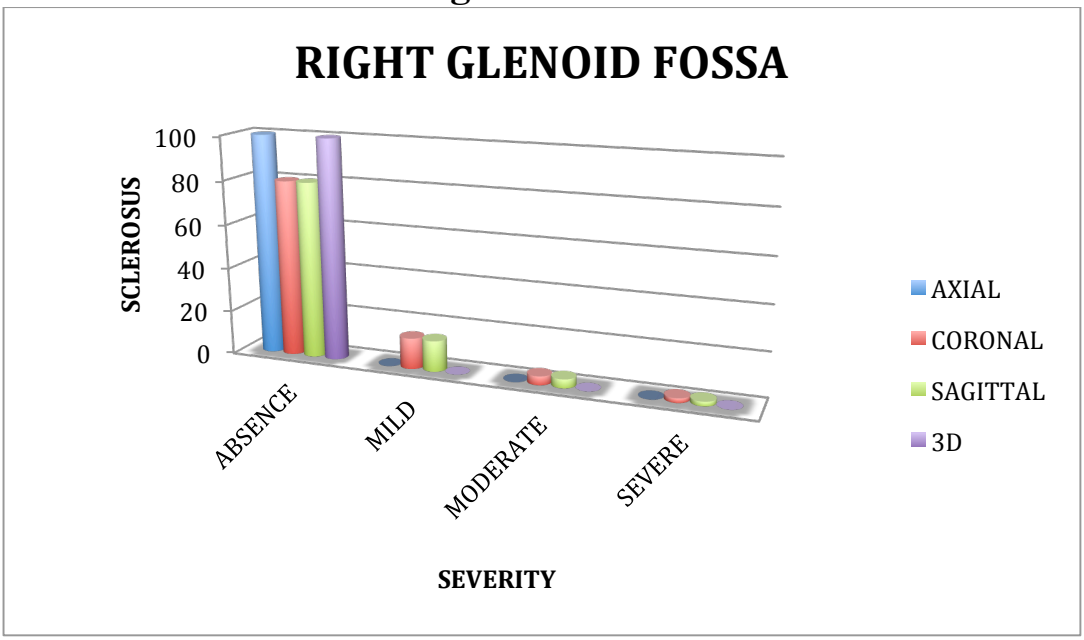


CHART 15- Distribution of severity of sclerosis in right glenoid fossa



RESULTS

Totally 50 cases of rheumatoid arthritis were included, having complaints of pain at TMJ region at rest or motion, joint sound such as crepitus and restricted mouth opening using clinical examination. These patients were then subjected to CBCT for both the TMJ regions (100 TMJ) for radiological assessment.

Out of 50 patients in the study, there were 47 (94%) females and 3 (6%) males as shown in **Chart 1**. A female predilection was observed. The mean age was found to be 45 years**.** Minimum age of the patient in the study was 25 and maximum age was 60 year. **Table 1** showing the age of distribution of the cases**. Table 2** showing the association of side of TMJ involvement with clinical parameters. 26% of the patients complained of pain in left TMJ at rest, while 60% complained of pain in left TMJ at motion. For right side joint, 16% patients presented with pain at rest and 56% presented with pain at motion. Crepitus was heard in 28% and 16% of the patients at left and right TMJ respectively. 20% of the patients complained for restricted mouth opening as shown in **table 3.**

CBCT was taken for both the left and right TMJ region. Radiological analysis was performed for 100 TMJ. Assessment was done for condylar process, glenoid fossa and joint space separately.

The patient was subjected to cone-beam computed tomography (CBCT) of the TMJ. **Table 4** shows evidence of various radiographic features of the osseous changes of the condyle. In axial section 21% of the condylar surface showed erosions as compared to 0% flattening, 8% sclerosis, 5% subchondral cyst and 6% osteophytes. This result was

found to be highly statistically significant ($p < 0.05$). In coronal section, 47% of the condylar surface showed erosion as compared to 14% flattening, 19% sclerosis, 10% subchondral cyst and 23% osteophytes. This result was found to be highly statistically significant ($p < 0.05$). In sagittal section, 42% of the condylar surface showed erosion as compared to 18% flattening, 13% sclerosis, 6% subchondral cyst and 24% osteophytes. This result was found to be highly statistically significant ($p < 0.05$). In 3D view, 50% of the condylar surface showed erosion as compared to 10% flattening, 0% sclerosis, 0% subchondral cyst and 26% osteophytes. This result was found to be highly statistically significant ($p < 0.05$).

Comparison of radiographic features based on clinical finding and Statistical significance was determined using the Mann-Whitney test. In **table 5**, when pain at rest was present at left TMJ and compared with erosion at left condyle, it results in p value of axial- 0.96, coronal- 0.80, sagittal- 0.54 and 3D view- 0.92. This result was found to be statistically not significant. Pain at left TMJ at rest compared with flattening at left condyle in axial, coronal, sagittal and 3D reconstructed view results in p value of axial- 1.0, coronal- 0.71, sagittal- 0.57 and 3D view- 0.98 and reported statistically not significant result.

Association of pain at motion at left TMJ with erosion of left condylar surface results in p value of axial- 0.70, coronal- 0.75, sagittal- 0.53 and 3D view- 0.97 and when compared with flattening results in p value of axial- 1.0, coronal- 0.90, sagittal- 0.78 and 3D view- 0.61. Both are found as statistically not significant.

Similar non- significant results obtained when comparison of pain at rest or

motion at left TMJ was made with radiographic feature of sclerosis and subchondral cysts in all the four sections of CBCT.

When both the clinical findings of pain at rest and motion at left TMJ was compared to radiographic feature of osteophyte at left condyle, it results in p value of 0.02 and 0.03 respectively, **which showed statistically significant values**. It means, presence of osteophyte was found to be significant in sagittal section of CBCT at left condylar surface, in subjects with no pain at rest and motion at left condyle.

Table 6 shows comparison of pain at rest and motion of left TMJ with erosion at right side condyle, it results in p value of axial- 0.63, coronal- 0.89, sagittal- 0.89 and 3D view- 0.64 in association with left TMJ pain at rest and axial- 1.0, coronal- 0.56, sagittal- 0.56 and 3D view- 0.90 in association with left TMJ pain at motion. This result was found to be statistically not significant. Pain at left TMJ at rest and motion gave p value of axial- 1.0, coronal- 0.96, sagittal- 0.86, 3D view- 0.43 and axial-1.0, coronal- 0.09, sagittal- 0.13 and 3D view- 0.24 when compared with flattening at right condyle and reported statistically not significant result.

Similar non-significant result was seen, when pain at rest and motion at left TMJ was correlated with sclerosis, subchondral cyst and osteophyte of other side condylar process on CBCT.

Table 7 shows pain at rest and motion at right TMJ comparison with radiographic features of other side (left) condyle in relation to erosion, flattening, sclerosis, subchondral cyst and osteophytes reported non significant result.

Table 8 depicts comparison of pain at right TMJ at rest and motion was done with bony changes of right side condylar surface. When pain at rest at right side TMJ compared to erosion on same side results in p value of axial- 0.70, coronal- 0.87, sagittal- 0.87 and 3D view- 0.68 for pain at rest and p value of axial- 0.67, coronal- 0.30, sagittal- 0.64 and 3D view- 0.69 for pain at motion. This result was found to be statistically not significant. While for flattening at right condylar surface, p value was axial- 1.0, coronal- 0.36, sagittal- 0.89 and 3D view- 0.53 for pain at rest at right TMJ as compared to p value of axial- 1.0, coronal- 0.19, sagittal- 0.45 and 3D view- 0.86 for pain at motion at right TMJ. For sclerosis at right condylar surface, p value came as axial- 0.61, coronal- 0.57, sagittal- 0.22 and 3D view- 1.0 for pain at rest at right TMJ and, axial- 0.42, coronal- 0.48, sagittal- 0.75 and 3D view- 1.0 for pain at motion of right TMJ. For subchondral cyst at right condyle, association with clinical features of pain at rest and motion at right side results in p value of axial- 0.66, coronal- 0.40, sagittal- 0.40, 3D view- 1.0, and axial- 0.37, coronal- 0.11, sagittal- 0.11, 3D view- 1.0 respectively. This result was found to be statistically not significant. Similar non significant result was found for radiographic assessment of osteophyte at right side in relation of right side pain at rest and motion.

Crepitus heard at left TMJ was compared with left side condylar imaging features on CBCT **(Table 9)**. Correlation with erosive changes at left condylar surface results in p value of axial- 0.09, coronal- 0.15, sagittal- 0.26 and 3D view- 0.28 respectively when crepitus heard at left TMJ, and p value of axial- 0.71, coronal- 0.44, sagittal- 0.55 and 3D view- 0.49, when crepitus heard at right TMJ. The result was statistically not significant. To find correlation with flattening on left condylar surface in all four sections of CBCT

results in p value of axial- 1.0, coronal- 0.15, sagittal- 0.11 and 3D view- 0.6 when crepitus heard at left TMJ, while p value of axial- 1.0, coronal- 0.54, sagittal- 0.46 and 3D view- 0.74 when crepitus heard at right TMJ. The result was found as statistically not significant. Similar non significant result was found when left and right TMJ crepitus was compared to left condylar imaging features. Crepitus heard at left TMJ was correlated to left condylar surface osseous changes. When sclerotic changes were assessed, results in p value of axial- 0.19, coronal- 0.91, sagittal- 0.37 and 3D view- 1.0. While crepitation at right TMJ, assessed for sclerotic changes in opposite side (left) of condyle, results in **p value of 0.04 in axial, 0.01 in coronal and 0.00 in sagittal showed highly significant result.**

TMJ assessment at right condyle was done in **table 10,** when crepitus heard at left and right TMJ. Bony changes like erosion, flattening, sclerosis and osteophyte in axial, coronal, sagittal and 3D reconstructed view respectively showed not significant result. While crepitus heard at right TMJ showed **p value of 0.02** when correlated to subchondral cyst at right condyle. This result was found to be statistically significant.

Recorded mouth opening of the patients was correlated with the erosion, flattening, sclerosis, subchondral cyst and osteophyte on left and right side condyle in **table 11**. The result was found to be statistically insignificant.

CHART 2 depicts severity of erosion in left side condyles. Of 50 left side condyles, absence of erosion was seen in axial- 78%, coronal- 46%, sagittal- 56% and 3D view - 44%. Mild erosion was seen as axial- 8%, coronal- 40%, sagittal- 30% and 3D view- 42% respectively and moderate erosion was seen as 14% in all sections of CBCT.

CHART 3 depicts severity of sclerosis in left side condyles. Absence of sclerosis was seen in axial- 92%, coronal- 80%, 86% and 3D view- 100%. Mild sclerosis was seen as axial- 6%, coronal- 18%, sagittal- 12% and 3D view- 0% respectively and moderate sclerois was seen as 2% in axial, coronal and sagittal section while 0% in 3D reconstructed view.

CHART 4 depicts evidence of subchondral cyst in left side condyles. Single subchondral cyst was seen in axial- 12%, coronal- 12%, sagittal- 4% and 3D view- 0% in respective sections while multiple subchondral cyst was seen in 2% of axial, coronal and sagittal section while 0% in 3D reconstructed view.

CHART 5 depicts severity of osteophytes in left side condyles. Mild osteophyte was present in axial- 2%, coronal- 18%, sagittal- 18% and 3D view- 20 of left condyle. While moderate osteophyte was present in 4% of axial section of condyle and 6% in remaining sections.

CHART 6 depicts severity of erosion in right side condyles. Mild erosion was seen as axial- 8%, coronal- 28%, sagittal- 28% and 3D view- 32% and moderate and severe erosion was seen as 4% and 8% in al sections of CBCT.

CHART 7 depicts severity of sclerosis in right side condyles. Mild sclerosis was seen as axial- 4%, coronal- 10%, sagittal- 4% in axial, coronal and sagittal section while 0% in 3D view and moderate sclerosis was seen in axial- 2%, coronal- 4%, sagittal- 4%, while 0% again in 3D view. Same frequency like moderate was seen for severe sclerosis.

CHART 8 depicts evidence of subchondral cyst in right side condyles. single

subchondral cyst was seen in 2% of axial and 4% in coronal as well as sagittal section in left condyle. While multiple subchondral cyst was seen in 2% of coronal and 2% of sagittal sections of right condyle.

CHART 9 depicts severity of osteophytes in right side condyles. mild osteophyte was present in axial- 6%, coronal- 16%, sagittal- 18% and 3D view- 20% in axial, coronal, sagittal and 3D reconstructed view of left condyle. While moderate osteophyte present in 2% of coronal, sagittal and 3D reconstructed view of condyle and severe osteophyte was present in 6% of condyle.

Table 12 shows evidence of various radiographic features of the osseous changes of the glenoid fossa. In axial section glenoid fossa showed no erosion, flattening and sclerosis. This result was found to be statistically non significant. In coronal section, glenoid fossa shows 10%- erosion, 23% flattening and 16% sclerosis. This result was found to be **statistically significant**. In sagittal section, 11% of the glenoid fossa showed erosion as compared to 28% flattening and 16% sclerosis. This result was found to be highly **statistically significant.**

CHART 10 shows severity of erosion in left side glenoid fossa. Of 50 left side glenoid fossa. Mild erosion was seen as 6% in every section other than axial, where no erosion was seen in any subject. Moderate erosion was seen as 2% in every section except axial.

CHART 11 shows severity of flattening in left side glenoid fossa. Mild flattening was seen as coronal- 8%, sagittal- 14%, while 4% in 3D view and moderate flattening was seen in coronal- 6% and sagittal- 6%.

CHART 12 shows severity of sclerosis in left side glenoid fossa. Mild sclerosis was seen as coronal- 8% and sagittal- 4% and moderate sclerosis was seen coronal- 8% and sagittal- 4%.

CHART 13 shows severity of erosion in right side glenoid fossa. Of 50 right side glenoid fossa. Mild erosion was seen as coronal- 8%, sagittal- 10% and 3D- 10%. Moderated erosion is seen in 2% of all sections except axial view. Severe erosion was also seen in 2% of all section sections while axial shows no erosion.

CHART 14 shows severity of flattening in right side glenoid fossa. Absence of flattening was seen in axial- 100%, coronal- 68%, sagittal- 66% and 3D view- 88%. Mild flattening was seen as coronal- 22%, sagittal- 22% and 3D view- 12% respectively and moderate flattening was seen as 8% in axial, coronal – 10% and sagittal- 10% of right side glenoid fossa. Severe flattening was seen in coronal- 2% and sagittal- 2%.

CHART 15 shows severity of sclerosis in right side glenoid fossa. no sclerosis was evident in axial and 3D view. Mild sclerosis was seen in coronal- 14% and sagittal- 14% and moderate sclerosis was seen coronal- 4% and sagittal- 4%. Severe sclerosis was seen in coronal – 2% and sagittal – 2%.

JOINT SPACE ASSESSMENT-

Out of 100 TMJ examined for joint space assessment in **TABLE -13**, 35% of them were having normal joint space whereas reduced joint space was seen in 65% of the TMJ.

TABLE 1: AGE DISTRIBUTION

	n	Range	Minimum	Maximum	Mean	Std. deviation
Age	50	35	25	60	45.2	9.8

TABLE 2: DISTRIBUTION OF SYMPTOMS

Clinical parameters	Side	Present	Absent	Total
Pain at rest	Left TMJ (n)	13	37	50
	Left TMJ (%)	26%	74%	100%
	Right TMJ (n)	8	42	50
	Right TMJ (%)	16%	84%	100%
Pain at motion	Left TMJ (n)	30	20	50
	Left TMJ (%)	60%	40%	100%
	Right TMJ (n)	28	22	50
	Right TMJ (%)	56%	44%	100%
Crepitus	Left TMJ	14	36	50
	Left TMJ (%)	28%	72%	100%
	Right TMJ	8	42	50
	Right TMJ (%)	16%	84%	100%

TABLE 3- DISTRIBUTION OF MOUTH OPENING

	NORMAL	RESTRICTED
MOUTH OPENING	80%	20%

TABLE 4- DISTRIBUTION OF OSSEOUS CHANGES IN CONDYLE

Radiographic parameters		Erosion	Flattening	Sclerosis	Subchondral cyst	Osteophyte	P val
I*	Absent n (n%)	79 (79%)	100 (100%)	92 (92%)	95 (95%)	94 (94%)	**0.00**
	Present n (n%)	21 (21%)	0 (0%)	8 (8%)	5 (5%)	6 (6%)	
II*	Absent n (n%)	53 (53%)	86 (86%)	81 (81%)	90 (90%)	77 (77%)	**0.00**
	Present n (n%)	47 (47%)	14 (14%)	19 (19%)	10 (10%)	23 (23%)	
III*	Absent n (n%)	58 (58%)	82 (82%)	87 (87%)	94 (94%)	76 (76%)	**0.00**
	Present n (n%)	42 (42%)	18 (18%)	13 (13%)	6 (6%)	24 (24%)	
IV*	Absent n (n%)	50 (50%)	90 (90%)	100 (100%)	100 (100%)	74 (74%)	**0.00**
	Present n (n%)	50 (50%)	10 (10%)	0 (0%)	0 (0%)	26 (26%)	

***I- AXIAL, II- CORONAL, III- SAGITTAL, IV- 3D**

TABLE 5- COMPARISON OF LEFT TMJ PAIN WITH LEFT CONDYLAR OSSEOUS CHANGES

	Left condylar osseous changes (P values)							
	Pain at rest (Left TMJ)				Pain at motion (left TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.96	0.80	0.54	0.92	0.70	0.75	0.53	0.97
Flattening	1.00	0.71	0.57	0.98	1.00	0.90	0.78	0.61
Sclerosis	0.94	0.79	0.90	1.00	0.51	0.45	0.49	1.00
Subchondral cyst	0.26	0.90	0.30	1.00	0.52	0.81	0.32	1.00
Osteophyte	0.29	0.82	**0.02**	0.68	0.32	0.38	**0.03**	0.06

TABLE 6- COMPARISON OF LEFT TMJ PAIN WITH RIGHT CONDYLAR OSSEOUS CHANGES

	Right condylar osseous changes (P values)							
	Pain at rest (Left TMJ)				Pain at motion (Left TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.63	0.89	0.89	0.64	1.00	0.56	0.56	0.90
Flattening	1.00	0.96	0.86	0.43	1.00	0.09	0.13	0.24
Sclerosis	0.25	0.58	0.58	1.00	0.67	0.76	0.15	1.00
Subchondral cyst	0.09	0.29	0.76	1.00	0.41	0.33	0.81	1.00
Osteophyte	0.76	0.50	0.40	0.31	0.81	0.27	0.89	0.60

TABLE 7- COMPARISON OF RIGHT TMJ PAIN WITH LEFT CONDYLAR OSSEOUS CHANGES

	Left condylar osseous changes (P values)							
	Pain at rest (right TMJ)				Pain at motion (right TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.86	0.80	0.71	0.72	0.94	0.47	0.71	0.63
Flattening	1.00	0.54	0.46	0.18	1.00	0.26	0.43	0.06
Sclerosis	0.66	0.78	0.46	1.00	0.41	0.73	0.91	1.00
Subchondral cyst	0.60	0.43	0.73	1.00	0.80	1.00	0.40	1.00
Osteophyte	0.73	0.22	0.63	0.18	0.39	0.18	0.06	0.10

TABLE 8- COMPARISON OF RIGHT TMJ PAIN WITH RIGHT CONDYLAR OSSEOUS CHANGES

	Right condylar osseous changes (P value)							
	Pain at rest (right TMJ)				Pain at motion (right TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.70	0.87	0.87	0.68	0.67	0.30	0.64	0.69
Flattening	1.00	0.36	0.89	0.53	1.00	0.19	0.45	0.86
Sclerosis	0.61	0.57	0.22	1.00	0.42	0.48	0.75	1.00
Subchondral cyst	0.66	0.40	0.40	1.00	0.37	0.11	0.11	1.00
Osteophyte	0.44	0.10	0.41	0.37	0.70	0.56	0.39	0.64

TABLE 9- COMPARISON OF CREPITATION WITH LEFT CONDYLAR OSSEOUS CHANGES

	Left condylar osseous changes (P value)							
	Crepitus (left TMJ)				Crepitus (right TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.09	0.15	0.26	0.28	0.71	0.44	0.55	0.49
Flattening	1.00	0.15	0.11	0.60	1.00	0.54	0.46	0.74
Sclerosis	0.19	0.91	0.37	1.00	**0.04**	**0.01**	**0.00**	1.00
Subchondral cyst	0.89	0.37	0.85	1.00	0.61	0.35	0.44	1.00
Osteophyte	0.27	**0.01**	0.28	0.06	0.38	0.31	0.31	0.38

TABLE 10 - COMPARISON OF CREPITATION WITH RIGHT CONDYLAR OSSEOUS CHANGES

	Right condylar osseous changes (P value)							
	Crepitus (left TMJ)				Crepitus (right TMJ)			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.53	0.30	0.70	0.46	0.70	0.35	0.35	0.24
Flattening	1.00	0.89	0.97	0.48	1.00	0.36	0.89	0.53
Sclerosis	0.89	0.69	0.20	1.00	0.61	0.57	0.96	1.00
Subchondral cyst	0.53	0.83	0.83	1.00	0.02	0.44	0.44	1.00
Osteophyte	0.83	0.11	0.32	0.24	0.44	0.10	0.08	0.07

TABLE 11- COMPARISON OF MOUTH OPENING WITH CONDYLAR OSSEOUS CHANGE

	Mouth opening (P value)							
	Left TMJ osseous changes				Right TMJ osseous changes			
	Axial	Coronal	Sagittal	3D	Axial	Coronal	Sagittal	3D
Erosion	0.82	0.71	0.72	0.62	0.38	0.47	0.47	0.67
Flattening	1.00	0.93	0.38	0.63	1.00	0.30	0.15	0.47
Sclerosis	0.30	0.37	0.15	1.00	0.30	0.85	0.82	1.00
Subchondral cyst	0.79	0.67	0.57	1.00	0.61	0.55	0.03	1.00
Osteophyte	0.37	0.23	0.06	0.18	0.37	0.86	0.62	0.75

TABLE 12- DISTRIBUTION OF JOINT SPACE

	LEFT SIDE	RIGHT SIDE	TOTAL
NORMAL JOINT SPACE	18%	17%	35%
REDUCED JOINT SPACE	32%	33%	65%

TABLE 13- DISTRIBUTION OF OSSEOUS CHANGES IN GLENOID FOSSA

Radiographic parameters		Erosion	Flattening	Sclerosis	P value
I*	Absent n (n%)	100(100%)	100(100%)	100(100%)	1.00
	Present n (n%)	0(0%)	0(0%)	0(0%)	
II*	Absent n (n%)	90(90%)	77(77%)	84(84%)	**0.05**
	Present n (n%)	10(10%)	23(23%)	16(16%)	
III*	Absent n (n%)	89(89%)	72(72%)	84 (84%)	**0.006**
	Present n (n%)	11(11%)	28(28%)	16 (16%)	
IV*	Absent n (n%)	89 (89%)	93(93%)	100(100%)	**0.004**
	Present n (n%)	11 (11%)	7(7%)	0(0%)	

***I- AXIAL, II- CORONAL, III- SAGITTAL, IV- 3D**

DISCUSSION-

Rheumatoid arthritis is an autoimmune disease of unknown cause, and has worldwide prevalence of 1%, affecting females more than males (3:1)[1]. Of the 50 patients in present study, there was increased prevalence of female 47 (94%), which was comparable to those published by other authors. In a study by **Linos A et al**[12] for prevalence of RA, prevalence rates for RA were 5.8/1000 for males and 13.4/1000 for females. The incidence was approximately double in women compared with that in men, in a study by **Gabriel SE et al**[16]**.** Another study by **Symmons DP et al (1994)**[15] showed the annual incidence rate of 36/100,000 for women and 14/100,000 for men.

RA is seen mainly in 35 and 55 years of age group[1]. The mean age in our study was found to be 45 years. Minimum age of the patient in the study was 25 and maximum age was 60 year. In a study documented by **Kurtoglu, et al**[19], mean age of RA was 46.56 years. In an analysis by **Symmons DP**[15] **et al,** RA was found to be rare in men aged under 45 years, and the incidence in women rose upto age 45 yrs. According to a data given in a study by **Yi-Chun Lin et al**[42], mean age was reported as 56.3 years.

In present study, rheumatoid arthritis patients with TMJ complaints were included. The symptoms taken into consideration were pain at TMJ region at rest or motion, joint sound such as crepitus and restricted mouth opening. **Yi-Chun Lin et al**[42] concluded that TMD is a frequent manifestation of RA, and approximately half of the cases of TMJ involvement in RA developed profound symptoms or joint abnormalities. In present study, 26% of the patients complained of pain in left TMJ at rest, while 60% complained of pain in left TMJ at motion. For right side joint, 16% patients presented

with pain at rest and 56% presented with pain at motion, clearly depicting that patients with pain at motion, is greater than patients with pain at rest. The major complaint as pain in patients with RA was also seen in studies by other authors like **F Ardic et al**[61] where 69.7 % of patients reported with pain complaint. In a study by **Abhijeet Deoghare et al**[62]**,** the most common finding in patients with RA was joint tenderness (70%), followed by joint crepitus (60%). In present study crepitus was heard in 28% and 16% of the patients at left and right TMJ respectively. Pain followed by crepitus, were the major symptoms seen in present study, and order was found to be similar with other studies. In a study by **C Kurtoglu et al**[19]**.** 9.25% of patients had pain in joints, and crepitations in 7.41% of patients. The result was contrary to study by **Yamakawa M et al**[41]**,** where 1.4% of patients with RA had unprovoked TMJ pain, 4.9% had pain on mouth opening, 9.2% had TMJ tenderness and crepitus in 35.9%, representing a significant excess occurrence of crepitus.

Clinical characteristic of restricted mouth opening in patients with RA was also considered in present study and 20% of the patients found to be complained for the same, which was found to be similar to a study by **F Ardic et al**[61], where 27.3% of patients with RA were having limited mouth opening. It was found to be less as compared to study by **Abhijeet Deoghare et al**[62]**,** where restricted mouth opening was seen in 60% of the subjects.

The patients were subjected to cone-beam computed tomography (CBCT) of the TMJ, to find out osseous changes to assess condylar process, glenoid fossa and joint space separately. Radiological analysis was performed for 100 joints and 84% of joints were found to have bony changes. **Abhijeet Deoghare et al**[62] detected radiological

findings in 88.8% of joints with RA in a study**.**

In a study by **Yi-Chun Lin et al[42],** 74.5% of RA patients had abnormal radiological findings. Few studies showed lesser radiographic involvement also, such as **Celiker R et al[44]** detected TMJ involvement by imaging techniques in 45% of subjects with RA.

Among all the radiographic features included for assessment of condylar process at the TMJ region, erosion was found to be in axial- 21%, coronal- 47%, sagittal- 42% and 3D view- 50%. This result was found to be **highly statistically significant,** suggestive of erosion as the major imaging finding in patients of rheumatoid arthritis as compared to other radiographic features such as osteophyte, flattening, sclerosis and subchondral cyst. **Goran W.Gynther et al (1991)[45]** concluded that no radiographic criterion pathognomonic for generalized osteoarthritis or rheumatoid arthritis, however, osteophytes, flattening of condyle or reduced joint space more in generalized osteoarthritis and erosions in condyle more in rheumatoid arthritis. Radiographically, erosion is seen as break in the cortical bone because of osteoclasts inducing factors, produced by synovial pannus, leading to increased resorption at pannus bone interface.(5,63) In a study by **Abhijeet Deoghare et al[62]**, predominant finding was erosion (85%) of the condyle in rheumatoid arthritis patients. Osteophyte was found to be second major radiological finding in present study. Osteophyte was found to axial- 6%, coronal- 23%, sagittal- 24% and 3D view- 26%. **Celiker R et al (1995)[44]** concluded erosion and osteophyte formation as the most frequent pathological signs in patients with rheumatoid arthritis.

In present study, when comparison is performed between clinical symptom of pain at rest or motion at left TMJ, with radiographic finding at same (left) condyle. This result was found to be statistically significant ($p < 0.05$), suggestive of presence of osteophytes without symptom of pain at rest or motion in rheumatoid arthritis patients. This statistically significant presence of osteophyte was seen in sagittal section of the CBCT, interpreting sagittal section as a view of choice to find out evidence of osteophyte. Joint instability is the main trigger for development of osteophyte. The cartilage of the condylar surface and the glenoid fossa undergo remodeling, when mechanical stress is beyond the adaptive capacity, by increasing the surface area to withstand loading forces. Calcium pyrophosphate crystal deposition has been stated to influence development of osteophyte. Osteophyte formation is seen to occur before the joint space narrowing and is found to be the major risk factor for arthritic condition development[64]. The significant presence of osteophyte in present study in subjects with no pain may be considered as an indicator for development of arthritic condition in that joint in future.

When crepitus at left TMJ was correlated with osseous changes at same condyle, results in $p < 0.05$ and found to be statistically significant, suggesting that osteophyte is more evident in joint with crepitation.

Sclerosis, an another imaging feature of rheumatoid arthritis was seen in axial- 8%, coronal- 19%, sagittal- 13% followed by flattening and subchondral cyst. A study by **Abhijeet Deoghare et al[62]**, sclerosis was predominant finding next to erosion. In present study when comparison was done between crepitation at right TMJ and osseous changes at left condyle in rheumatoid arthritis patients. The comparison showed highly significant

result, indicating that crepitus in one joint can be a reason for sclerotic changes in other joint.

TMJ acts as a single unit and any disturbance in function on one side can cause altered function on the opposite side[65]. Abnormal loading on joint will lead to micro damage, high bone remodeling and vascular invasion in the subchondral bone and led to sclerosis in the subchondral bone[66]. Sclerosis is considered to be a sign of healing of joint and reflects a stage of bone repair[62].

Assessment of glenoid fossa is also performed in this study. The evidence of flattening of glenoid fossa at TMJ region is found to be significant, suggesting flattening as a major imaging finding in glenoid fossa of patients with rheumatoid arthritis and, sagittal followed by coronal view are the section of choices for diagnosing flattening at the glenoid fossa. Our result was found to be contradictory with few studies. **Abhijeet Deoghare et al[62],** stated that condyle can be affected in both osteoarthritis and rheumatoid arthritis but glenoid fossa primarily involve the glenoid fossa. They found out erosion of glenoid fossa as a major finding at the glenoid fossa. A study by **Kshar Avinash et al[67],** found erosion followed by flattening as the most common radiographic change in patients with rheumatoid arthritis.

In this study, out of 100 TMJ examined for joint space assessment, 35% of them were having normal joint space whereas reduced joint space was seen in 65% of the TMJ, signifying narrowing of joint space in patients with rheumatoid arthritis. **Celiker R et al (1995)[44]** in a study on subjects having rheumatoid arthritis with TMJ involvement found decreased joint space among frequent pathological signs at TMJ region.

CONCLUSION AND SUMMARY

This study was conducted to evaluate the CBCT features in rheumatoid arthritis patients with TMJ involvement. This study included 50 patients, of which 47 were females and 3 were males showing female predominance. The patients in the present study fell into the age group ranging from 25 to 60 years. The mean age was found to be 45 years. CBCT was taken for 100 TMJ and assessment was done for condyle, glenoid fossa and joint space separately. Clinical findings of the patients are correlated with the CBCT imaging features. In present study, 26% of the patients complained of pain in left TMJ at rest while 60% complained of pain in left TMJ at motion. For right side joint, 16% patients presented with pain at rest and 56% presented with pain at motion, clearly depicting that patients with pain at motion is greater in number than patients with pain at rest. Pain followed by crepitus and reduced mouth opening are the symptoms seen in present study.

Erosion was the major imaging finding in patients, followed by osteophytes. When comparison is performed between clinical symptoms and imaging features, statistically significant presence of osteophyte was seen in sagittal section of the CBCT to the joints of the patients having absence of pain, interpreting sagittal section as a view of choice to find out evidence of osteophyte, as well as osteophyte presence is an indicator of development of arthritic conditions.

When crepitus at left TMJ was correlated with osseous changes at same condyle results in $p < 0.05$, suggesting that osteophyte is more evident and significant in joint with crepitation. Moreover, when crepitus at right TMJ was correlated with osseous

changes at opposite side condyle, the comparison showed highly significant result, indicating that crepitus in one joint can be a reason for sclerotic changes in opposite joint.

The study depicts flattening as a major imaging finding in glenoid fossa of patients with rheumatoid arthritis, and sagittal followed by coronal view are the section of choices for diagnosing flattening at the glenoid fossa. Reduced joint space was seen in 65% of the TMJ, signifying narrowing of joint space in patients with rheumatoid arthritis.

Limitations in the present study lie on the duration of the illness, which is not compared to clinical findings and imaging features.

CBCT was found to be accurate in diagnosing all the arthritic changes such as erosion, flattening, sclerosis, subchondral cyst and osteophytes. CBCT was also found to be equally effective in depicting arthritic changes at glenoid fossa and assessing the dimensions of joint space.

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TAMIL NADU GOVRNMENT DENTAL COLLEGE & HOSPITAL, CHENNAI – 3.

TELEPHONE : 044-253403343
FAX: 044- 25300681

date : 24/09/2015

Ref No: R.C No.0430/DE/2015 dated 27.01.2015, O/O Principal, TNGDC
Sub: IEC review of the research proposals,

Title of the work: Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography

Principal Investigator: Dr. Priyanka
II Yr. M.D.S., Student.

Department : Department of Oral Medicine and Radiology
Tamil Nadu Govt. Dental College & Hospital , Chennai-3

Thank you for submitting your research proposal , which was considered at the Institutional Ethics Committee meeting held on 02-07-2015, at TN Govt. Dental College and the documents related to the study referred above were discussed and the modifications done as suggested and reported to us have been reviewed.

The decision of the members of the committee , the secretary and the Chairperson IEC of TN Govt. Dental College is here under:

Approved	Approved and advised to proceed with the study
Approved with suggestions	----------
Revision	----------
Rejected	----------

The principal investigators and their team are advised to adhere the guide lines given below:

11. You should get detailed informed consent from the patients / participants and maintain confidentiality.
12. You should carry out the work without affecting regular work and without extra expenditure to the Institution or the Government.
13. You should inform the IEC, in case of any change of study procedure, site, and investigating guide.
14. You should not deviate from the area of work for which you have applied for ethical clearance.
15. You should inform the IEC immediately in case of any adverse events or serious adverse reactions. You should abide to the rules and regulations of the institution(s) .
16. You should complete the work within specific period and if any extension of time is required, you should apply for permission again to do the work.
17. You should submit the summary of the work to the ethical committee every 3 months and on completion of the work.
18. You should not claim any kind of funds from the institution for doing the work or on completion/ or for any kind of compensations.
19. The members of the IEC have the right to monitor the work without prior intimation.
20. Your work should be carried out under the direct supervision of the guide/ Professor.

MEMBER SECRETARY,
INSTITUTIONAL ETHICS COMMITTEE
Tamil Nadu Govt. Dental College & Hospital
Chennai

CHAIRPERSON
INSTITUTIONAL ETHICS COMMITTEE
Tamil Nadu Govt. Dental College & Hospital
Chennai

PATIENT INFORMATION SHEET

Title of the study "Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography"

Name of research institution- Tamilnadu Government Dental College and Hospital, Chennai – 3

Purpose of the study

The purpose of the study is to evaluate osseous changes in rheumatoid arthritis patients with temporomandibular joint involvement (TMD) using Cone Beam Computed Tomography.

Procedures

Patient selection followed by obtaining through history and informed consent. Complete clinical examination using diagnostic instruments. CBCT for patients confirmed with rheumatoid arthritis with TMJ involvement. Asseesment of osseous changes in TMJ.

Benefits of participation

Early diagnosis for intervention to reduce severity of disease.

Participant's rights

Taking part in this study is voluntary. Patients are free to decide whether to participate in this study or to withdraw at any time; patients decision will not result in any loss of benefits to which you are otherwise entitled.The results of this special study may be intimated to patient at the end of the study period.

Risk of participation

Participants are selected based on proper inclusion and exclusion criteria. So, risk of participation is negligibe or atleast manageable.

Confidentiality

The identity of the patients participating in the research will be kept confidential throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Compensation - Nil

Contacts for queries related to the study: Dr. Priyanka, II year PG student,

Department of Oral Medicine and Radiology,

Tamilnadu Government Dental College,

Chennai 600 003 (Ph.- 8939495025)

ஆராய்ச்சி பற்றிய தகவல் படிவம்

கூம்பு வடிவ கணினி வரைவி பரிசோதனை மூலம் முடக்குவாத நோயாளிகளின் தாடை மூட்டில் வடிவ மாற்றத்தைக் கண்டறிதல் என்ற ஆய்வை தமிழ்நாடு அரசு பல் மருத்துவக் கல்லூரி மருத்துவமனைக்கு வரும் நோயாளிகளிடம் நடத்த உள்ளோம். அதற்காக மேற்கண் நோயாளிகளை தேர்வு செய்கிறோம்.

நோயாளிகள் பற்றிய குறிப்புகள் ஆராய்ச்சி முடியும் வரை ரகசியமாக பாதுகாக்கப்படும். இந்த ஆராய்ச்சியை வெளியிடும்போது நோயாளிகளின் தனிப்பட்ட விவரங்கள் எதுவும் பாதிக்கப்படமாட்டாது.

இந்த ஆராய்ச்சியில் பங்குபெறுவது நோயாளிகளின் தனிப்பட்ட விருப்பம். மேலும் நோயாளிகள் இந்த ஆராய்ச்சியிலிருந்து எப்போது வேண்டுமானாலும் விலகிக்கொள்ளலாம். நோயாளியின் இந்த முடிவினால் அவருக்கோ அல்லது ஆராய்ச்சியாளருக்கோ எந்தவித பாதிப்பும் கிடையாது.

இந்த சோதனை மேம்படுத்தப்பட்ட ஊடுகதிர் நகல் ஆய்வு மூலம் தாடை மூட்டு நோயை விரைவாக கண்டறியவும், அதன் மூலம் அதை குணப்படுத்தவும் மறைமுகமாக உதவுகிறது.

இந்த ஆராய்ச்சியின் முடிவுகள் நோயாளிகளுக்கு ஆராய்ச்சியின் இடையிலோ அல்லது முடிவிலோ தெரிவிக்கப்படும். இதில் ஏதேனும், பின் விளைவுகள் ஏற்பட்டால் அதை சரிசெய்ய சிகிச்சையளிக்க தகுந்த உதவிகள் செய்யப்படும்.

ஆய்வாளரின் கையொப்பம் **பங்கேற்பாளர் கையொப்பம்**

தேதி :

இடம் :

ஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு

கூம்பு வடிவ கணினி வரைவி பரிசோதனை மூலம் முடக்குவாத நோயாளிகளின் தாடை மூட்டில் வடிவ மாற்றத்தைக் கண்டறிதல்

ஆராய்ச்சி நிலையம் : அரசு பல் மருத்துவக் கல்லூரி
சென்னை

பங்கு பெறுபவரின் பெயர் :

பங்கு பெறுபவரின் எண் :

பங்கு பெறுபவரின் பிறந்த தேதி : ..... / ..... / .....
தேதி மாதம் வருடம்

இந்த ஆய்வு சம்பந்தமாக நான் மேலே கூறப்பட்ட தகவல் படிவத்தை முழுமையாக படித்துப் பார்த்தேன் என்று உறுதி கூறுகிறேன்.

நான் இது தொடர்பான அனைத்து கேள்விகளுக்கும் நிறைவான பதில்கள் பெறப்பட்டேன்.

இந்த ஆய்வின் எனது பங்கு தன்னிச்சையானது என்றும் எந்த நேரத்திலும் இந்த ஆய்வில் இருந்து சட்ட உரிமைகள் பாதிக்கப்படாமல் விலகிக் கொள்ள சம்மதிக்கிறேன்.

இந்த சோதனை மேம்படுத்தப்பட்ட ஊடுகதிர் நகல் ஆய்வு மூலம் தாடை மூட்டு நோயை விரைவாக கண்டறியவும், அதன் மூலம் அதை குணப்படுத்தவும் மறைமுகமாக உதவுகிறது என்பதை புரிந்துகொண்டேன்.

மருத்துவ ஆய்வு அதிகாரிகள், எனது சிகிச்சை தொடர்பான பதிவேடுகளை பார்வையிடவும் எந்த நேரத்திலும், ஆய்வில் இருந்து நான் விலகினாலும் பார்வையிட சம்மதிக்கிறேன். எனது அடையாள குறிப்புகள் மூன்றாவது நபருக்கு தெரிவிக்கப்படமாட்டாது என்று புரிந்து கொண்டேன்.

இந்த ஆய்வு அறிக்கைகளை பயன்படுத்தவும், வெளியிடவும், நான் சம்மதிக்கிறேன். ஆய்வாளர் எனது மருத்துவக் குறிப்புகளை வெளியிட தடையாக இருக்கமாட்டேன் என உண்மையாக சம்மதிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ..... இடம் ..... தேதி .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் ..... இடம் ..... தேதி .....
ஆய்வாளரின் பெயர் .....

Annexure: AF 06/004/01.0
Template for Informed Consent Form

Informed Consent Form

"Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography"

Participant ID No:

"I have read the foregoing information, or it has been read to me. I have had the opportunity to ask questions about it and any questions I have asked have been answered to my satisfaction. I consent voluntarily to participate as a participant in this study and understand that I have the right to withdraw from the study at any time without in any way it affecting my further medical care."

Date	Name of the participant	Signature/thumb impression of the participant

[The literate witness selected by the participant must sign the informed consent form. The witness should not have any relationship with the research team; If the participant doesn't want to disclose his / her participation details to others, in view of respecting the wishes of the participant, he / she can be allowed to waive from the witness procedure (This is applicable to literate participant ONLY). This should be documented by the study staff by getting signature from the prospective participant]

_____

_____

—

"I have witnessed the accurate reading of the consent form to the potential participant and the individual has had opportunity to ask questions. I confirm that the individual has given consent freely"

Date	Name of the witness	Signature of the witness

Date	Name of the interviewer	Signature of the interviewer

DEPARTMENT OF ORAL MEDICINE AND RADIOLOGY

TAMIL NADU GOVT. DENTAL COLLEGE & HOSPITAL, CHENNAI -3

CASE PROFORMA

ASSESSMENT OF TMJ MORPHOLOGICAL CHANGES IN RHEUMATOID ARTHRITIS PATIENTS USING CONE BEAM COMPUTED TOMOGRAPH

Date: Serial no:

Name: O.P No:

Age/Sex:

Address:

Phone no:

Occupation: Income:

Religion:

Centre: Department of Oral Medicine And Radiology,

Tamil Nadu Govt Dental College & Hospital, Chennai -3

Presenting complaint with duration:

Past medical history:

Past Surgical history:

Past dental history:

Personal history:

A) Diet:

B) Teeth cleansing habits:
- Cleaning aids used:
- Frequency :

C) Smoking habit:
- Material used:
- Frequency :
- Duration of the habit:

D) Chewing habit:
- Material used:
- Frequency :
- Duration of the habit:

E) Other habits (alcohol, snuff):

Marital status:

Menstrual History:

Family history:

CLINICAL EXAMINATION

Extraoral Examination:

Facial asymmetry

Temporomandibular joint Examination-

Pain at rest-

Pain at motion-

Joint sound-

Intraoral examination:

Mouth opening(interincisal distance):

Size and Shape of mouth:

Jaw movements:

- Teeth:
- Gingiva:
- Alveolar mucosa
- Labial and buccal mucosa:
- Hard palate:
- Soft Palate:
- Pillar of fauces and Tonsils:
- Tongue:
- Floor of the mouth:
- Retromolartrigone:

Provisional diagnosis:

Investigations:

1) Laboratory investigations:

   A) Blood:

      RBC Count:

      Total WBC count:

      Differential count: P L E Bleeding time:

      Haemoglobin %: Clotting time:

      Peripheral smear:

      Erythrocyte sedimentation rate:

Clinical diagnosis:

2) Radiological investigations:

   CBCT findings-

Inferences:

Signature of PG student

Signature of Guide

Date:

TRIPARTITE AGREEMENT

This agreement herein after the "Agreement" is entered into on this day ----------between the Tamil Nadu Government Dental College and Hospital represented by its **Principal** having address at Tamil Nadu Government Dental College and Hospital, Chennai – 600 003, (hereafter referred to as, 'the college')

And

Prof Dr. S JAYACHANDRAN aged 52 years working as **Professor and HOD** in Department of Oral Medicine and Radiology at the Tamil Nadu Government Dental College, having residence address at A.M. 16, TNHB Quarters,Todhunter Nagar, Saidapet, Chennai - 600 015.Tamil Nadu. (Herein after referred to as 'Principal Investigator')

And **Dr. PRIYANKA** aged 26 years currently studying as **Post Graduate student** in Department of Oral Medicine and Radiology, Tamil Nadu Government Dental College, House no. 802, sector 3, Urban Estate, Kurukshetra, Haryana. Pin no-136118 . (herein after referred to as the 'PG and co- Investigator').

Whereas the PG student as part of her curriculum undertakes this research on "**Assessment of TMJ morphological changes in rheumatoid arthritis patients using cone beam computed tomography"** for which purpose the Guide shall act as Principal investigator and the college shall provide the requisite infrastructure based on availability and also provide facility to the PG student as to the extent possible as a Co- investigator.

Whereas the parties, by this agreement have mutually agreed to the various issues including in particular the copyright and confidentiality issues that arise in this regard.

Now this agreement witnessed as follows

1. The parties agree that all the Research material and ownership therein shall become the vested right of the college, including in particular all the copyright in the literature including the study, research and all other related papers.
2. To the extent that the college has the legal right to do go, shall grant to licence or assign the copyright so vested with it for medical and/or commercial usage of interested persons/ entities subject to a reasonable terms/ conditions including royalty as deemed by the college.
3. The royalty so received by the college shall be shared equally by all the three parties.
4. The Co-investigator and Principal Investigator shall under no circumstances deal with the copyright, Confidential information and know – how – generated during the course of research/study in any manner whatsoever, while shall sole west with the college.

5. The Co-investigator and Principal Investigator undertake not to divulge (or) cause to be divulged any of the Confidential information or, know – how to anyone in any manner whatsoever and for any purpose without the express written consent of the college.
6. All expenses pertaining to the research shall be decided upon by the Principal investigator/ Co-investigator or borne sole by the PG student (Co-investigator)
7. The college shall provide all infrastructure and access facilities within and in other institutes to the extent possible. This includes patient interactions, introductory letters, recommendation letters and such other acts requires in this regard.
8. The Principal Investigator shall suitably guide Co-investigator the Student Right from selection of the Research Topic and Area till its completion. However the selection and conduct of research, topic and area of research by the student researcher under guidance from the Co-Investigator shall be subject to the prior approval, recommendations and comments of the Ethical Committee of the College constituted for the purpose.
9. It is agreed that as regards other aspects not covered under this agreement, but which pertain to the research undertaken by the Co-investigator, under the guidance from the Principal Investigator, the decision of the college may be binding and final.
10. If any dispute arises as to the matters related or connected to this agreement herein, it shall be referred to arbitration in accordance with the provisions of the Arbitration and Conciliation Act, 1996.

In witness whereof the parties hereinabove mentioned have on this day month and year herein above mentioned set their hands to this agreement in the presence of the following two witnesses.

College represented by its **Principal** **PG Student**

Witnesses **Student Guide**

1.

2.

S.No	NAME	AFE/SEX	Pain Left TMJ Rest	Pain Right TMJ Rest	Pain Left TMJ Motion	Pain Right TMJ Motion	Crepitus Left	Crepitus Right	Mouth opening
1.	AMBIKA	25/F	1	1	1	1	1	0	1
2.	AMSA	53/F	0	0	1	0	0	0	0
3.	AMSAVALLI	38/F	0	0	1	0	0	0	0
4.	ANURADHA	40/F	1	0	1	1	0	0	1
5.	AZHAGU	57/F	0	1	1	1	1	0	0
6.	BANUPRIA	43/F	0	0	0	0	0	0	0
7.	BHARATHI	37/F	1	0	1	1	0	1	0
8.	BHARATHI	36/F	1	0	1	1	0	0	1
9.	BHAVANI	53/F	0	0	0	0	0	0	0
10.	BHUVANI	58/F	0	0	1	0	0	0	0
11.	DEVIKA	49/F	0	0	0	1	0	0	0
12.	DEVI	53/F	0	0	0	1	0	0	0
13.	ELAMMAL	55/F	0	1	1	1	1	0	1
14.	KALIAPERUMAL	50/F	0	0	0	0	0	0	0
15.	KAMALA	56/F	0	0	0	1	1	1	0
16.	KAMALAVALI	33/F	0	0	0	1	0	0	0
17.	KANAGA	57/F	0	0	1	1	0	0	1
18.	KOMALAVA	29/F	1	0	1	1	0	0	0
19.	KRISHNA	60/F	0	0	1	1	0	0	1
20.	LAKSHMI	48/F	0	0	1	1	1	0	0
21.	MALLIGA	55/F	0	0	1	0	0	0	0
22.	MALLIGA	40/F	0	0	0	0	1	0	0
23.	MALLIKA	55/F	1	1	1	1	1	0	1
24.	MALLIKA	43/F	0	0	0	0	1	1	0

25.	PAPPA	33/F	0	0	1	0	0	1	0
26.	PETER	54/M	0	0	0	0	0	0	0
27.	PONGODAI	33/F	0	0	1	1	0	0	0
28.	PONNI	33/F	0	0	0	0	0	0	0
29.	PUNITHA	36/F	0	1	1	1	1	0	0
30.	PUSHPA	47/F	0	0	1	0	0	0	0
31.	RADHA	50/F	0	0	1	1	0	0	0
32.	RAJESHWARI	34/F	0	0	1	1	0	0	0
33.	RAJESHWARI	34/F	1	0	1	1	0	1	0
34.	RANJITHAM	45/F	1	0	1	1	1	0	0
35.	REVATHI	38/F	1	0	1	1	0	0	1
36.	RUBY	40/F	0	0	1	1	0	1	0
37.	SANTHA	50/F	0	0	0	0	0	0	0
38.	SANTHA	56/F	0	1	1	1	1	0	0
39.	SATHYA	53/F	1	0	0	0	0	0	0
40.	SEETHA	25/F	0	0	0	0	0	0	0
41.	SHANKAR	60/M	0	0	0	0	0	0	0
42.	SHANTHI	49/F	0	0	0	0	1	0	0
43.	SHEELA	45/F	1	0	1	1	0	0	1
44.	SIVAMANI	43/F	0	0	0	0	0	0	0
45.	SUDHA	60/F	0	0	0	0	0	0	0
46.	SUNDAR	40/M	0	1	1	1	0	0	0
47.	SUPARNA	36/F	1	0	1	1	1	0	0
48.	SUSILA	60/F	0	0	0	0	0	1	0
49.	VIJAYA	42/F	0	0	0	0	0	1	0
50.	VIJAYLA	52/F	1	1	1	1	1	0	1

S.NO.	NAME	AGE/SEX	SIDE	EROSION (CONDYLE)				FLATTENING (CONDYLE)				SCLEROSIS (CONDYLE)				SUBCHONDRAL CYST (CONDYLE)				OSTEOPHYTE (CONDYLE)			
				I	II	III	IV	I	II	III	IV	I	II	III	IV	I	II	III	IV	I	II	III	IV
1	AMBIKA	25/f	L	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	2	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
2	AMSA	53/f	L	0	0	0	0	0	3	3	3	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0
3	AMSAVALLI	38/f	L	0	0	0	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	1	1
4	ANURADHA	40/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
5	AZHAGU	57/f	L	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
6	BANUPRIA	43/f	L	0	0	0	0	0	1	1	1	0	1	1	0	1	1	0	0	0	0	0	0
			R	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	1	0	1
7	BHARATHI	37/f	L	0	0	0	0	0	0	0	0	1	1	1	0	1	1	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	1	1	0	0	1	0	0	0	0	0	0	0
8	BHARATHI	36/f	L	2	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9	BHAVANI	53/f	L	1	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1
10	BHUVANI	58/f	L	0	1	1	1	0	0	0	0	0	0	0	0	1	0	0	0	0	1	1	1
			R	3	3	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
11	DEVIKA	49/f	L	2	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
			R	0	1	1	1	0	0	0	0	2	2	2	0	0	2	2	0	0	0	0	0
12	DEVI	53/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1
			R	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2	2
13	ELAMMAL	55/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

			R	2	2	2	2	0	0	0	0	0	1	1	0	0	1	1	0	0	0	1	1
14	KALIAPERUMAL	50/f	L	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
15	KAMALA	56/f	L	0	1	1	1	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
16	KAMALAVALI	33/f	L	0	1	1	1	0	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	1	1	1
17	KANAGA	57/f	L	0	1	1	1	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0
			R	3	3	3	3	0	0	0	0	0	1	0	0	0	0	0	0	0	3	3	3
18	KOMALAVALI	29/f	L	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0	0	1	0	1
			R	3	3	3	3	0	0	0	0	0	1	0	0	0	0	0	0	0	1	1	1
19	KRISHNA	60/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1
20	LAKSHMI	48/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
21	MALLIGA	55/f	L	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2	2
			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
22	MALLIGA	40/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
23	MALLIKA	55/f	L	0	1	1	1	0	0	0	0	0	1	0	0	1	1	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
24	MULLIKA	43/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
25	PAPPA	33/f	L	0	1	1	1	0	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0
			R	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
26	PETER	54/m	L	2	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1
			R	0	0	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
27	PONGODAI	33/f	L	0	1	0	1	0	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0

			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1
28	PONNI	33/f	L	0	1	0	1	0	0	0	0	0	0	0	0	0	2	2	0	0	0	0	0
			R	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	3	3	3
29	PUNITHA	36/f	L	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
30	PUSHPA	47/f	L	0	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0
31	RADHA	50/f	L	2	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
32	RAJESHWARI	34/f	L	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	1	1	1	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
33	RAJESHWARI	34/f	L	0	1	0	1	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0
34	RANJITHAM	45/f	L	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1
35	REVATHI	38/f	L	0	0	0	0	0	2	2	2	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
36	RUBY	40/f	L	2	2	2	2	0	0	0	0	2	2	2	0	0	0	0	0	0	1	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
37	SANTHA	50/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
38	SANTHA	56/f	L	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	1	1	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0
39	SATHYA	53/f	L	1	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	1	0	1
			R	0	0	0	0	0	0	0	0	3	3	3	0	0	0	0	0	0	0	0	0
40	SEETHA	25/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
41	SHANKAR	60/m	L	0	1	1	1	0	1	1	1	0	0	0	0	0	0	0	0	2	2	2	2

			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	1	1	1	1
42	SHANTHI	49/f	L	0	0	1	1	0	0	0	0	0	1	0	0	0	0	1	0	0	0	1	0
			R	0	0	1	1	0	0	0	0	0	3	3	0	0	0	0	0	0	0	0	0
43	SHEELA	45/f	L	0	1	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
44	SIVAMANI	43/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
45	SUDHA	60/f	L	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
			R	3	3	3	3	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
46	SUNDAR	40/m	L	2	2	2	2	0	0	0	0	0	1	0	0	0	1	0	0	0	0	1	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
47	SUPARNA	36/f	L	0	1	0	1	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	1	1	1	0	0	0	0	0	0	0	0	0	0	0	0
48	SUSILA	60/f	L	2	2	2	2	0	0	0	0	0	0	0	0	0	1	0	0	0	1	1	1
			R	1	1	1	1	0	0	0	0	0	2	2	0	0	0	0	0	0	0	0	0
49	VIJAYA	42/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	2	2	2	2
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
50	VIJAYLA	52/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0

***I- AXIAL, II- CORONAL, III- SAGITTAL, IV- 3D**

S.NO.	NAME	A/S	SIDE	EROSION (GLENOID FOSSA)				FLATTENING (GLENOID FOSSA)				SCLEROSIS (GLENOID FOSSA)				Joint Space
				I	II	III	IV	I	II	III	IV	I	II	III	IV	
1	AMBIKA	25/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
2	AMSA	53/f	L	0	0	0	0	0	1	1	0	0	0	0	0	1
			R	0	0	0	0	0	1	1	0	0	0	0	0	1
3	AMSAVALLI	38/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	2	2	0	0	0	0	0	1
4	ANURADHA	40/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
5	AZHAGU	57/f	L	0	0	0	0	0	0	1	0	0	0	0	0	0
			R	0	0	0	0	0	0	1	0	0	0	0	0	0
6	BANUPRIA	43/f	L	0	0	0	0	0	3	3	0	0	0	0	0	0
			R	0	0	0	0	0	0	1	0	0	0	0	0	0
7	BHARATHI	37/f	L	0	1	1	1	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
8	BHARATHI	36/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	1	1	1	0	0	0	0	0	0	0	0	1
9	BHAVANI	53/f	L	0	1	1	1	0	1	1	0	0	0	0	0	1
			R	0	0	0	0	0	1	1	0	0	0	0	0	0
10	BHUVANI	58/f	L	0	0	0	0	0	1	1	0	0	0	0	0	0
			R	0	2	2	2	0	0	0	0	0	0	0	0	0
11	DEVIKA	49/f	L	0	0	0	0	0	2	2	1	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
12	DEVI	53/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
13	ELAMMAL	55/f	L	0	0	0	0	0	0	0	0	0	1	1	0	1
			R	0	0	0	0	0	2	2	1	0	0	0	0	0

14	KALIAPERUMAL	50/f	L	0	1	1	1	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	3	3	1	0	0	0	0	1
15	KAMALA	56/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
16	KAMALAVALI	33/f	L	0	0	0	0	0	0	1	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
17	KANAGA	57/f	L	0	0	0	0	0	2	2	1	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	2	2	0	1
18	KOMALAVALI	29/f	L	0	0	0	0	0	2	2	1	0	0	0	0	1
			R	0	1	1	1	0	0	0	0	0	2	2	0	1
19	KRISHNA	60/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
20	LAKSHMI	48/f	L	0	0	0	0	0	0	0	0	0	2	2	0	1
			R	0	0	0	0	0	0	0	0	0	1	1	0	0
21	MALLIGA	55/f	L	0	0	0	0	0	0	0	0	0	1	1	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
22	MALLIGA	40/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
23	MALLIKA	55/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
24	MULLIKA	43/f	L	0	2	2	2	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	1	1	0	0
25	PAPPA	33/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
26	PETER	54/m	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
27	PONGODAI	33/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1

28	PONNI	33/f	L	0	0	1	1	0	0	2	1	0	1	1	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
29	PUNITHA	36/f	L	0	0	0	0	0	1	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	1	1	0	0
30	PUSHPA	47/f	L	0	0	0	0	0	1	1	1	0	0	0	0	0
			R	0	0	0	0	0	1	1	0	0	0	0	0	1
31	RADHA	50/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
32	RAJESHWARI	34/f	L	0	0	0	0	0	0	1	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
33	RAJESHWARI	34/f	L	0	1	1	1	0	1	1	0	0	0	0	0	1
			R	0	1	1	1	0	0	1	0	0	0	0	0	1
34	RANJITHAM	45/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
35	REVATHI	38/f	L	0	0	0	0	0	1	1	0	0	1	1	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
36	RUBY	40/f	L	0	3	3	3	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
37	SANTHA	50/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
38	SANTHA	56/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	1	1	0	1
39	SATHYA	53/f	L	0	0	0	0	0	0	0	0	0	1	1	0	0
			R	0	0	0	0	0	2	2	0	0	0	0	0	1
40	SEETHA	25/f	L	0	0	0	0	0	0	0	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
41	SHANKAR	60/m	L	0	0	0	0	0	1	1	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1

42	SHANTHI	49/f	L	0	0	0	0	0	0	0	0	0	2	2	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
43	SHEELA	45/f	L	0	0	0	0	0	1	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	0
44	SIVAMANI	43/f	L	0	0	0	0	0	0	0	0	0	1	1	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
45	SUDHA	60/f	L	0	0	0	0	0	1	1	0	0	0	0	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
46	SUNDAR	40/m	L	0	0	0	0	0	0	0	0	0	1	1	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
47	SUPARNA	36/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	1	1	0	0	0	0	0	0
48	SUSILA	60/f	L	0	0	0	0	0	0	0	0	0	3	3	0	1
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
49	VIJAYA	42/f	L	0	0	0	0	0	2	2	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1
50	VIJAYLA	52/f	L	0	0	0	0	0	0	0	0	0	0	0	0	0
			R	0	0	0	0	0	0	0	0	0	0	0	0	1

***I- AXIAL, II- CORONAL, III- SAGITTAL, IV- 3D**